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INTRA-ABDOMINAL HERNIA AS A CAUSE OF INTESTINAL OBSTRUCTION WITH SPECIAL REFERENCE TO DUODENAL FOSSAE

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INTRA-ABDOMINAL hernias are rare and are most frequently found incidentally at postmortem, but when they do produce symptoms, they are usually the symptoms of acute intestinal obstruction. These symptoms may be and usually are the first warning of the presence of an intra-abdominal hernia. When confronted with a patient with intestinal obstruction of a mechanical type and when there is no apparent reason for the obstruction, it is well to keep this rather rare condition in mind. Without a thorough examination, obscure conditions such as small nodules in the femoral regions can be overlooked until surgical exploration demonstrates them to be causes of obstruction.[13] When these types of cases are ruled out, and when there is no history of previous intra-abdominal surgery, internal hernias are the most frequent causes of mechanical obstruction.[18]

Up to 1939,[10] 467 well documented cases of intra-abdominal hernia had been reported. This, we believe, is not a true representation of the frequency of the condition, for many surgeons who have had the experience of operating in one case have not reported it. It is with this in mind that this subject is discussed and a case of hernia into the paraduodenal fossa of Landzert causing acute intestinal obstruction is reported. Special emphasis will be placed on hernias into the fossae about the duodenum.

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In a report of the largest single series of such cases, Mayo et al[17] reviewed 39 cases of intra-abdominal hernia and found 18 (46 per cent) to be primary. Of these 18 cases, 8 were "duodenal," 6 being on the left side, and 2 on the right side. In Hannsman and Morton's[10] series of 467 collected cases, approximately 60 per cent were located about the duodenum and when mesenteric hernias are added, and these happen usually in the vicinity of the duodenum, this percentage was increased to approximately 70 per cent. From these statistics, the importance of the region about the duodenum is clearly brought out. Many bizarre types of intra-abdominal hernia such as hernia into the suspensory ligament of the ovary,[22] herniation of the splenic flexure of the colon behind the spleen,[20] herniation through the foramen of Winslow in which the bowel returned to the general abdominal cavity through the gastrohepatic omentum,[7] herniation into the broad ligament as a primary hernia,[15, 9] herniation into the lesser peritoneal sac,[11, 12, 25] and many others have been reported,[14, 19, 21, 23, 24, 26] but these types are extremely rare, and because of the frequency of the location about the duodenum, exploration in patients suspected of having intra-abdominal hernia should be performed with this in mind.

There are 7 possible places where herniation and strangulation of the bowel can occur within the abdomen. These are in the fossae about the duodenum, including the foramen of Winslow, around the cecum, around the sigmoid, in the supra-vesical region, through the diaphragm, in exaggerations of normal mucosal folds such as the broad ligaments and congenital defects, and through the omentum. This list does not include intestinal protrusions secondary to previous intra-abdominal surgery especially gastro-enterostomy and uterine suspension operations such as the Baldy-Webster type, to inflammatory conditions, and conditions resulting from trauma. Obstruction with no apparent predisposing cause is the type under discussion, and when a careful history is obtained from the patient, intra-abdominal operations, inflammatory processes, and trauma should be ruled out as a cause of the lesion present. In addition to the anatomically recognized places where hernias occur, one must always keep in mind the ever present possibility of a peritoneal anomaly due to malrotation of the bowel or malformation of the peritoneal attachments of the bowels. Because of the extensive literature on diaphragmatic hernias, this will only be mentioned as a possible site of intestinal obstruction and will not be discussed further.

Four sites which have fossae and/or recesses where intra-abdominal hernias may occur have one or more anatomic areas into which bowel may herniate. These are the paraduodenal area, the

area around the cecum, around the sigmoid, and in the supravesical region.

Moynihan[16] describes 9 fossae about the duodenum and this does not include the foramen of Winslow. Four of these are in-

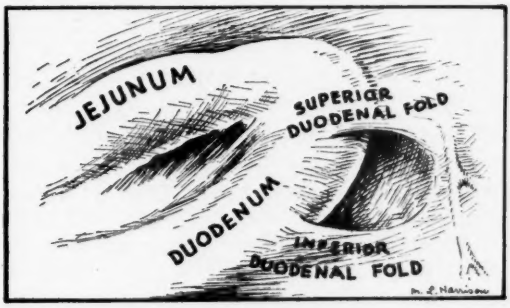

Fig. 1. An anatomic drawing showing the orifice of the most frequent paraduodenal fossae and the inferior mesenteric vein in the anterior leaf of the orifice of the fossa of Landzert along with the superior and inferior duodenal folds.

frequently observed and are considered unimportant. The 5 commonly observed fossae are: (1) the inferior duodenal fossa, which is a recess in the peritoneum alongside the lower and external part of the ascending limb of the duodenum and whose ostium is to the left and its apex points downward and to the right. The anterior surface of its entrance is made up by the inferior duodenojejunal fold. This structure does not contain any blood vessels of importance; (2) the superior duodenal fossa whose ostium is directed inferiorly and whose recess is located along the upper and external part of the ascending portion of the duodenum. The superior duodenojejunal fold forms the anterior wall of its orifice; (3) the paraduodenal fossa of Landzert which lies to the left of the terminal portion of the duodenum. It is thought that this fossa is formed by a raising of the peritoneum by the inferior mesenteric vein and the left colic artery and has been described as "the mesentery" of that vein. Its ostium points downward and to the right and its pouch points somewhat upward and to the left. The fold which makes up the anterior wall of its opening carries the inferior mesenteric vein and may contain the left colic artery. This fold may unite with the folds forming the anterior walls of the superior and inferior duodenal fossae and thereby cause some confusion as to the exact limits of each fossa. Figure 1 is an anatomic drawing which illustrates in a somewhat exaggerated manner the usual location of the orifices of the more frequent paraduodenal fossae and demonstrates the inferior mesenteric vein in the fold of peritoneum overlying the orifice

to the paraduodenal fossa of Landzert. The presence of the inferior mesenteric vein and the left colic artery in the anterior wall of this fossa makes it necessary to exercise great care in dividing the neck of the sac in cases of strangulation of bowel in this fossa; (4) the mesentericoparietal fossa which is, at times, called the parajejunal fossa is described by Moynihan[16] as being located most frequently in the mesojejunum immediately behind the superior mesenteric artery. Its orifice looks mainly to the left, and its blind pouch to the right and downward. Its anterior wall carries the superior mesenteric artery, and its posterior wall is made up of the peritoneum over the lumbar vertebrae. Here again it is important to be extremely careful in dividing the neck of the sac, for the superior mesenteric artery could easily be severed with disastrous results. This fossa is infrequent in that Desjardins in examining 100 bodies found only one such fossa. Another fossa which is frequently misunderstood as to its exact nature is the fossa of Waldeyer which is actually a combination of the inferior and superior duodenal fossae into one fossa. The inferior fossa is the most frequently present fossa and the superior duodenal fossa is next in frequency. When present, the paraduodenal fossa of Landzert is the longest of the duodenal fossae. In reviewing the anatomy of these fossae frequent discrepancies are found. Some state that the parajejunal fossa, fossa of Waldyer, and mesenticoparietal fossa are synonymous. After careful anatomic research, the descriptions as given above are considered to be correct.

In the region of the cecum, there are 4 fossae or recesses. The superior ileocecal or, as it is sometimes called, the ileocolic recess is located in the angle between the ileum and the ascending colon and its anterior boundary is made up by a reflection of peritoneum from the anterior aspect of the ascending colon to the anterior surface of the mesentery of the terminal ileum and carries the anterior cecal branch of the ileocolic artery. The inferior ileocecal recess occupies a position between the ileocecal fold anteriorly and the mesoappendix posteriorly. The retrocecal fossa is located below the cecum and has the avascular retrocecal fold as an anterior boundary of its ostium as this fold extends from the ascending colon and cecum across the lower part of the right paracolic gutter to the peritoneum of the right iliac fossa. The paracecal recess is located lateral to the cecum in the right lateral gutter and has the cecal fold as the anterior wall of its ostium. Hernias into these fossa and recesses are infrequent; however, if all cases found were reported, the incidence probably would be much greater than the published figures would have one believe.

There is only one fossa in the region of the sigmoid and that is

the intersigmoid recess which is a small funnel-shaped pouch at the apex of the vertical and horizontal roots of the ascending mesocolon.

Warvi and Orr[29] reviewed all of the available literature on hernias in the supravesical region and point out that there are 5 possible routes or directions in which hernias into these fossae may travel. Two of these routes lead to external type hernias whereas the other 3 would be intra-abdominal. The internal hernias arising in this fossa could extend into the prevesical space, to the fundus of the bladder, and to the paravesical space. They point out that this type hernia is not nearly so rare as previously believed.

Several theories as to the etiology of "duodenal hernias" have been advanced. Andrews[1] refused to accept the then prevalent view that to have these hernias, fossae about the duodenojejunal junction must be present, and states that "duodenal hernia is a congenital anamoly due to imprisonment of the small intestine beneath the mesentery of the developing colon." This occurred secondary to abnormalities in rotation of the bowel. He pointed out that if rotation of the umbilical loop is not carried to completion a right duodenal hernia would result and considered left duodenal hernia simply as a result of a more pronounced variation from normal in this same process of rotation. This congenital anamoly would result in the superior mesenteric and the iliocolic arteries lying in the anterior leaf of the neck of the sac of right duodenal hernias and the inferior mesenteric artery would lie in the free edge of the neck of the sac of left duodenal hernias and the vein would lie above the artery and thereby complete the vascular arch of Treitz. Cogswell and Thomas[4] accepted Andrews' explanation of the formation of these hernias and reviewed again the embryology of intestinal rotation and pointed out how abnormalities of rotation could result in this type hernia. Callander et al[3] took issue with this explanation and stated, from a careful study of one of their cases of left duodenal hernia, that the hernia resulted from an invagination of an area of the descending mesocolon medial to the ascending branch of the left colic artery and the inferior mesenteric vein by the jejunoileum while the descending colon was mobile and had not yet fused with the primative posterior parietal peritoneum. Biermann[2] reported a case in which he had operated prior to the formation of a "duodenal hernia" and stated that at the first operation he found no evidence of the presence of any abnormality. This would indicate that at least all of the hernias cannot be regarded as congenital. Moynihan[16] expressed the belief that an increase in intra-abdominal pressure initiated the formation of these hernias provided the proper fossae were present and as a result of this increase in pres-

sure a loop of bowel would become engaged in the fossa resulting in the hernias. It is easily conceivable how, once a loop of bowel was engaged in a fossa, it could, through distention, produce a "gas-trap" mechanism and pull more bowel into the fossa through a small aperture. The fact that these fossa are found at the anatomy table empty of bowel would further indicate that the mechanism of congenital formation of these hernias is not a necessity.

The symptoms of intra-abdominal hernia vary from peculiar mild abdominal pain in cases in which the bowel is not obstructed or strangulated to those of fully developed intestinal obstruction. The patient to be presented had symptoms of acute intestinal obstruction but the abdominal pain was present for 2 days before complete obstipation developed. On physical examination in these cases, the abdomen is usually distended and somewhat tender throughout. Cases are described in which mases were palpable but none was present in our case. On x-ray examination, using a flat film of the abdomen, the usual signs of intestinal obstruction were present and Exner[8] and Hollenberg[12] have called attention to a clumped appearance of the small bowel in the epigastrium "as if they were contained in a sac," and state that this is the characteristic x-ray appearance of paraduodenal intra-abdominal lesions. In the case to be described the entire colon was greatly distended and there was in the mid-abdomen a group of dilated small bowel which were not in the usual step-ladder arrangement. This probably should have directed our attention to the pathology present but the large bowel distention and the frequency of colonic obstruction in the left side of the colon by malignant lesions resulted in a preoperative diagnosis of colonic obstruction probably due to a malignancy in the sigmoid or recto-sigmoid. Wangensteen[28] stated that the distended colon might have resulted from an incomplete obstruction being present for a time before becoming complete. In our case, the patient had bowel movements for 2 days after the development of symptoms, and when examined rectally, there was a small amount of blood-tinged liquid and some gas expelled.

We should like to emphasize that these lesions are rare and that they should not be confused with other lesions, for we have had a case since the one being reported that resulted in some embarrassment in that the 14 year old colored male had chronic obstruction and at the first operation, it was thought that he had an intra-abdominal hernia, but when he did not get well following operation and his obstruction persisted, a second operation revealed a carcinoma of the splenic flexure causing the obstruction. In reviewing 335 cases of intestinal obstruction McIver found only 3 cases of intra-abdominal hernia.

Report of Case

W. W. Y., a 70 year old white male, entered the hospital complaining of severe pain in the epigastrium of 4 days' duration, obstipation of approximately 60 hours, frequent vomiting during 4 days, hiccoughs of 48 hours' duration and abdominal distention for 48 hours. His illness had begun suddenly and had been characterized by sharp severe epigastric pains. These subsided only to recur in approximately 12 hours. From this time forward, the pain had been persistent. He had begun vomiting soon after the onset of the persistent pain. At the beginning, the vomitus was only slightly colored but soon became brownish black. It was never fecal in odor. His abdomen had begun to be distended approximately 48 hours prior to admission. This was concommitant with obstipation. Hiccoughs had begun 2 days prior to admission.

Fig. 2. Flat film of the abdomen showing greatly dilated colon and segment of small bowel in the mid-abdomen.

Prior to this illness, he had had frequent "sour stomachs" and gas but no abdominal pain or distention. During the past 2 years, he had had some substernal and high epigastric distress associated with dyspnea and "irregular heart beat." His present complaint was entirely different from anything he had experienced previously.

On physical examination his pulse was 64 and regular and his blood pressure was 160/90. His respiratory rate was 20 per minute and of a grunting character. He was obese and acutely ill. The only positive findings were limited to the abdomen and this was markedly distended and tender throughout with some rebound tenderness. No masses were palpable.

Rectal examination was negative for intrinsic pathology and on withdrawal of the examining finger a small amount of blood-tinged fluid was present and a small amount of flatus followed.

Routine laboratory examination of the blood and urine was negative except for a white blood count of 13,800 and a slight shift to the left in the hemogram. His N.P.N. was 40 and his blood sugar was 120 mg. per cent. Flat film of his abdomen (fig. 2) showed the colon to be greatly distended from the cecum

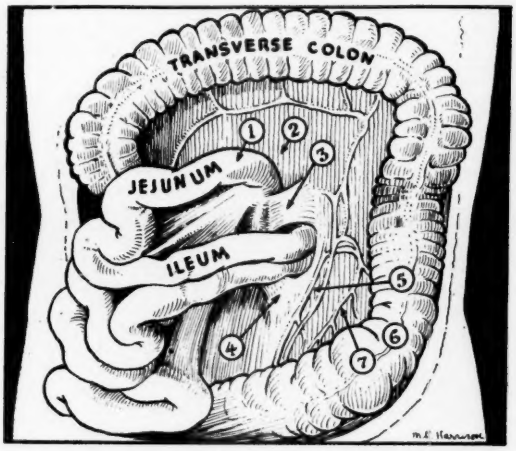

Fig. 3. An anatomic drawing illustrating the herniation of the ileum into the paraduodenal fossa of Landzert. The dilatation of the small bowel is not illustrated in order that the hernia may be better presented. (1) ileum; (2) transverse mesocolon; (3) superior duodenal fold; (4) inferior duodenal fold; (5) inferior mesenteric artery and vein; (6) descending colon; (7) inferior colic artery.

to the sigmoid and a group of distended small bowel in the mid-abdomen but not typical step-ladder effect. There appeared to be no fluid present.

A preoperative diagnosis of colonic obstruction in the sigmoid, probably on the basis of tumor, was made.

Intravenous fluids, gastric suction, and blood typing were started immediately. Following hydration and the procurement of compatible blood he was operated. Preparation took approximately 12 hours and during this time his distention was slightly improved.

At operation the colon was found greatly distended and it was thought that the preoperative diagnosis would be proved correct, but on thorough search of the entire colon no lesion could be found. This was so disappointing that the results by simple palpation would not be accepted and a large colon tube was passed into the left colon and the colon was again palpated along the course of the tube. A large amount of gas was expelled through the tube but still no lesion could be found. The small bowel was distended in its upper portion but the ileum near the cecum was flat. The collapsed bowel was followed and it was found to pass under a peritoneal fold in the paraduodenal area. Figure 3 is a drawing of the lesion found. The distention of the small bowel was not pictured in order that the hernia could be better illustrated. The fold contained a large vein and the herniated bowel was found to pass toward the left and under the descending colon. The hernia was easily reduced by simple traction and peristalsis was noted to cross the involved portion of small bowel and to fill the collapsed small bowel. The bowel wall was deep

reddish-blue and glistening. It was considered that the bowel was viable and the abdomen was closed in layers using interrupted cotton sutures. No attempt was made to close the ostium of the fossa. The patient was given 1000 c.c. of blood during and immediately following surgery.

On his return to bed he was given oxygen and his transfusion was continued. The colon tube which had been put in at operation was left in. He had a mild reaction to his transfusion. His fever began to rise, dyspnea became severe, and on examination he was found to have a massive collapse of the left lung. He was aspirated, given pressure oxygen, large doses of penicillin, and the gastric suction continued, but none of the treatment was of any avail and the patient died approximately 20 hours after surgery.

The treatment of these lesions is the same as that of any intestinal obstruction. Especial attention should be called to the anatomic arrangement about the orifice of the fossa for it would be a tragedy blindly to enlarge the neck of the sac and in so doing sever the inferior mesenteric vein in all cases and, in many, the left colic artery. Our case was easy in that simple traction reduced the hernia but when this is not true, we recommend opening the sac over the dilated bowel and deflating the bowel following Wangensteen's aseptic technic. This will make it possible to reduce the hernia.

SUMMARY

A case of intestinal obstruction produced by herniation of the ileum into the paraduodenal fossa of Lanzert has been presented, along with the anatomic details of the arrangement of the more prevalent places in the abdomen where intra-abdominal hernia can occur. The contents of the anterior leaf of the orifice of the fossa has been stressed and attention called to the disaster which might follow blind enlarging of the neck of the hernial sac. Though this condition is rare, it should not be overlooked as a cause of obscure intestinal obstruction. Doubt in the congenital origin of all these hernias has been expressed. The treatment of this condition is that of the associated intestinal obstruction.

BIBLIOGRAPHY

1. Andrews, E.: Duodenal Hernia: A Misnomer, Surg., Gynec. & Obst. *37*:740-750, 1923.
2. Biermann, W. J.: Hernia Into the Duodenojejunal Fossa (Hernia of Treitz); Report of 3 Cases, J. Internat. Coll. Surgeons *8*:521-528 (Nov.-Dec.) 1945.
3. Callender, C. L.; Rusk, G. Y., and Nemir, A.: Left Duodenal Hernia, Surg. Gynec. & Obst. *60*:1052-1071, 1935.
4. Cogswell, H. D., and Thomas, C. A.: Right Paraduodenal Hernia, Ann. Surg. *114*:1035-1041, 1941.
5. Copenhaver, N. H.: Intra-abdominal Hernias, Arch. Surg. *7*:322-346, 1923.
6. Dewis, J. W., and Miller, R. H.: Hernia Through the Foramen of Winslow; Report of a Case with Reference to 33 Other Cases Collected from the Literature, Surg. Gynec. & Obst. *45*:95-102, 1927.
7. Edwards, H., and Stewart, W.: Hernia Through the Foramen of Winslow Emerging Through the Gastro-Hepatic Omentum, Brit. M. J. *1*:343-345 (Mar. 20) 1943.

8. Exner, F. B.: The Roentgen Diagnosis of Right Paraduodenal Hernia, Am. J. Roentgenol *29*:585, 1933.
9. Goode, T. V., and Newbern, W. R.: Intestinal Obstruction Associated with Defects in the Broad Ligaments of the Uterus, Am. J. Surg. *65*:127-132 (July) 1944.
10. Hansmann, G. H., and Morton, S. A.: Intra-abdominal Hernia: Report of a Case and Review of the Literature, Arch. Surg. *39*:973-986 (Dec.) 1939.
11. Haxton, H. A.: Hernia of Ceacum Into Lesser Sac of Peritoneum Complicated by Volvulus, Brit. M. J. *2*:792 (Dec. 16) 1944.
12. Hollenberg, M. S.: Radiographic Diagnosis of Hernia Into Lesser Peritoneal Sac Through the Foramen of Winslow, Surgery *18*:498-502 (Oct.) 1945.
13. Jones, T. E., and Kehm, R. W.: Obscure Incarcerated or Strangulated Hernia as a Cause of Intestinal Obstruction, Cleveland Clin. Quart. *13*:8-10 (Jan.) 1946.
14. Langley, G. F.: Strangulated Internal Hernia; Report of Cases of Strangulation in the Fossa Iliaco-subfascialis and Through the Foramen of Winslow, Brit. J. Surg. *23*:119-127 (July) 1935.
15. Masson, J. C., and Atkinson, W.: Hernia Into Broad Ligament and Remarks on Other Intra-abdominal Hernias, Am. J. Obst. & Gynec.. *28*:731 (Nov.) 1934.
16. Moynihan, B. G. A.: On Retroperitoneal Hernia in: The Anatomy and Surgery of the Peritoneal Fossa, London, Bailliere, Tindall, and Cox, 1899.
17. Mayo, C. W.; Stalker, L. K., and Miller, J. M.: Intra-abdominal Hernia—Review of 39 Cases in Which Treatment Was Surgical, Ann. Surg. *114*:875 (Nov.) 1941.
18. Miller, J. M.: Intra-abdominal Hernias as a cause of Intestinal Obstruction, Proc. Staff. Meet., Mayo Clin. *15*:359 (June 5) 1940.
19. Mitchell, G. F.: Acute Intestinal Obstruction in a Baby 15 Months Old; Hernia Through the Foramen of Winslow, Brit. J. Surg. *26*:648-649 (Jan.) 1939.
20. Paul, L. W., and McAneny, T. B.: Herniation of the Spleenic Flexure of the Colon Behind the Spleen, Radiology *36*:230-232 (Feb.) 1941.
21. Pulrang, S.: Disastrous Sequelae to the Webster-Baldy Operation, Am. J. Surg. *65*:281-285 (Aug.) 1944.
22. Shepard, V. D., and Waugh, J. M.: Hernia into Suspensory Ligament of the Ovary Producing Intestinal Obstruction, Proc. Staff Meet., Mayo Clin. *17*:126-128 (Feb. 25) 1942.
23. Stalker, L. K.: Intra-abdominal Hernia with Acute Intestinal Obstruction, New York State J. Med. *45*:2307-2308 (Nov. 1) 1945.
24. Steinke, C. R.: Internal Hernia, Arch. Surg. *25*:909-925 (Nov.) 1932.
25. Tanner, M. C.: Hernia Traversing the Lesser Sac, Brit. J. Surg. *33*:184-186 (Oct.) 1945.
26. Ullman, A.: Hernia Through the Foramen of Winslow, Surg., Gynec. & Obst. *38*:225-233 (Feb.) 1924.
27. Wangensteen, O. H.: New Operative Techniques in the Management of Bowel Obstruction, Aseptic Decompressive Suction Enterotomy, Aseptic Enterotomy for Removal of Obstructing Gall Stone, and Operative Correction of Noxrotation, Surg., Gynec. & Obst. *75*:675-692 (Dec.) 1942.
28. Wangensteen, O. H.: Personal Communication to the authors.
29. Warvi, W. M., and Orr, T. G.: Internal Supravesical Hernia, Surgery *8*:312-325, 1940.

ACROMIOCLAVICULAR JOINT INJURIES

Pathology and Operative Treatment

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THE acromioclavicular joint is a simple, spheroidal diarthrosis. Its capsule is somewhat lax in all positions of the joint which has incongruent articular surfaces, thick cartilages and in some instances the presence of an articular disc. These, together with a sufficiently loose capsule, give this joint freedom of movement on many axes, permitting angular movements and rotation of the scapula on the end of the clavicle. There are no ordinary actions in which the scapula moves on a fixed clavicle, or vice versa; the two bones, bound together by their connecting ligaments, move in unison. The axes of the articular surface are directed backward and obliquely so that the clavicular facet tends to rest upon that of the acromion. The capsule is weak, thickened by superior and inferior acromioclavicular ligaments. This joint has a sliding anterior posterior movement so that when the shoulder is thrust forward the angle between the clavicle and acromion becomes smaller. The coracoclavicular ligament is widely held to be the source of greatest stability and much of the weight of the upper extremity is conveyed to the clavicle by this ligament. The clavicle keeps the arm and shoulder from collapsing on the chest. (The coracoacromial ligament also contributes to the strength of the region, forming a strong arch above the shoulder anterior to the acromioclavicular joint.) Most writers state that without the coracoclavicular ligament the ligaments of the joint itself could not maintain the articular surfaces in apposition. This ligament limits the clavicle backward, forward or directly upward from the acromion. The degree of obliquity with which the clavicular facet rests against and is supported by the acromion is another factor for stability. This prevents downward displacement of the clavicle and also explains why a downward force on the acromion tears the supporting ligaments by thrusting it under the clavicle. Movement in the acromioclavicular joint allows the glenoid fossa a little forward and backward motion. When motion in the acromioclavicular joint is restricted, the incidence of dislocation of the head of the humerus is logically increased. If the coracoclavicular ligaments are intact, the joint may be resected for disease or ankylosis without disability.

Two more cases of this condition have been seen since submission of the paper. One was operated 8 months ago and has normal or nearly so and completely painless shoulder. The other refused operation and has a painful arthritic acromioclavicular joint.

Classifications of anatomic variations in the acromioclavicular joint in 100 random roentgenograms of the shoulder: (Urist)

Description of Joint	*Per Cent*
1. The articular surface of the clavicle overrides the articular surface of the acromion	49
2. The articular surfaces of the acromion and clavicle are nearly vertical and lie in the same plane	27
3. The inferior margin of the articular surface of the clavicle underrides the superior margin of the acromion	3
4. The articular surfaces are incongruent, and the clavicle overlies the acromion	9
5. The articular surfaces are incongruent and are not in contact at any point	6
6. The articular surfaces are incongruent, and the inferior margin of the clavicle underrides the superior margin of the acromion	6

Some twenty-five or thirty methods of repair have been advocated for disruption of this joint. This is good evidence as to the failure of solution of the problem. Conservative therapy is based on some modification of the brachioclavicular sling, using a harness, plaster or various straps. Recommended treatment in the operative field varies from fusion to fascial repair and much mention is made of approximation of articular surfaces in spite of known incongruence anatomically. Reconstruction by fascia fails[8] because the new fascial ligaments fail to maintain reduction or because of persistent pain interpreted as arthritic changes in the joint. Mumford[40] resected the acromioclavicular joint in 4 cases of incomplete separation with relief. This is a very pertinent observation. We are in agreement with Urist[50] who demonstrated that complete dislocation of the acromioclavicular joint may occur with or without gross coracoclavicular ligament rupture. The normal excursion of the conoid and trapezoid ligaments permits dislocation when the joint alone is involved. Balanced muscle action by the trapezius and deltoid is probably the greatest single factor in maintaining proper apposition of joint surfaces. Howard[28] points out that a great range of glenohumeral and scapulothoracic movements is probable and is the reason subluxations do not occur more frequently. In adults clavicle dislocations are far more frequent in proportion to fractures than in children.

The essential point of restoration of joint function is controlled motion. The acromioclavicular joint, a diarthrodial joint, is constructed for motion. Mollier[39] gives the joint ranges as:

Acromioclavicular axes:	Vertical	15°
	Sagittal	19°
	Frontal	29°
Sternoclavicular:	Vertical	37°
	Sagittal	20°
	Horizontal	22°

He demonstrated that for every change in position of scapula, there is corresponding change in the position of the clavicle reflected by movement at these two joints and if either or both should be fixed or immobile, the total range of shoulder motion would be markedly impaired.

Twenty-six cases are reported on, all of which have been followed for more than one year. All gave a definite history of injury. Twenty-four were seen in the U. S. Naval Hospital, New Orleans, Louisiana, and 2 have been private patients since then. Age varied from 19 to 64 years. This latter was the only woman of the series and also one of the 2 non-operated cases. Only 4 were seen primarily (within 2 days), 6 within 4 weeks past injury, and the rest from 30 days to 22 months past injury. Only 12 (46 per cent) showed coracoclavicular ligament rupture, 2 had marked stretching of these ligaments and one had ruptured the trapezoid element only. Two had been operated previously, a figure 8 wire being passed through the acromioclavicular joint for fixation. These joints showed extreme damage.

Microscopic sections were made of 20 acromioclavicular joints and all show the same basic findings in varying stages of progress. This was essentially exuberant proliferation of young fibrous tissue from organized hemorrhage into the joint, fragmentation and degeneration of cartilage and overgrowth of cartilage by fibrous tissue. Also it was noted grossly that frequently the entire articular surface of either clavicle or acromion, but especially clavicle, would be undermined by creeping granulomatous tissue and practically detached. Loose bodies and cartilage flakes were also found. These findings corresponded with and substantiated correlated x-ray findings of arthritis of the joint. Roentgenographic arthritic changes were seen in 19 (about 78 per cent) cases, the earliest stigmata being suspected in 2 cases respectively 2 and 3 weeks after injury. Those not having such changes were operated electively (in the latter part of the series) within one week after injury with the exception of one case seen 4 weeks after trauma. It is thought that

the vertical type joint shows arthritis radiographically best and quickest and this would seem logical. Thorndyke and Quigley[48] believe that perhaps aseptic necrosis occurs to the ends of the clavicle and acromion. They think it may be a complication which

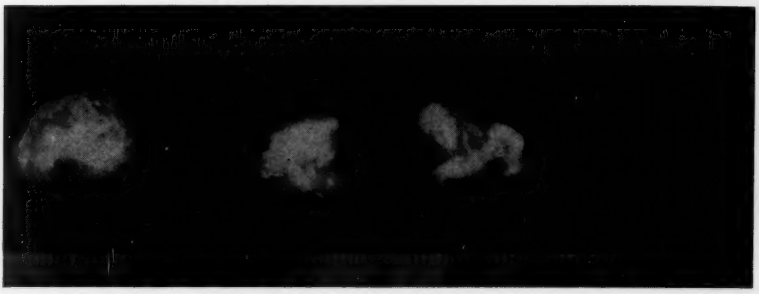

Fig. 1—Characteristic gross specimens of injured acromioclavicular joints.

heals with immobilization in 3 weeks or so but this seems doubtful in light of the above findings. Also arthritis of this joint is a frequent x-ray finding.

The operative treatment devised was first thought of for relief of demonstrable and disabling arthritis of the acromioclavicular joint in the more chronic cases. Later it seemed obvious and inevitable that a painful arthritic shoulder would develop and the procedure was done early as the method of choice. Also previous personal as well as recorded experience had been quite disappointing no matter what type therapy was used. The Roberts[43] approach is used routinely. At operation about one half (12) of the cases were found to show healing processes in the coracoclavicular ligaments varying from dense cicatricial repair to calcification and, in one case, even ossification. The articular cartilages are excised back to bone and as much exuberant granulation tissue as possible removed. An attempt is made to repair the capsule but this is not requisite. A hole is drilled in the clavicle in line with the coracoid and either another one put in it or the fixation wire passed about the coracoid process. The dislocation is reduced and held so by tightening a fairly heavy stainless steel wire (#26) which is doubled. The deltoid is reattached. A Velpeau-like support is used for about 2 weeks, shifting then to a sling. At about 4 weeks active unresisted exercises are started and increased progressively. The purpose of this procedure is, of course, to relieve a painful joint, securing a fibrous union only which will allow almost normal motion and at the same time provide for stability. Meanwhile, the coracoclavicular ligaments and especially the deltoid and trapezius muscles are allowed

to regain normal tone and relationship. All Naval personnel save one discharged for a coincident head injury were returned to full duty and remained at duty. The average postoperative time was just under 40 days. The two longest convalescences were 52 and 56

Fig. 2—Close up view of characteristic specimen of injured acromioclavicular joint.

days, one because of postoperative pneumonia, the other because of an unexplained footdrop. The one civilian operated on went back to his normal occupation as a painter in 60 days. The 2 patients not operated upon have continued to have painful and weak shoulders with limited use. It is not claimed that these shoulders have been restored to normal but this does seem to represent a definite improvement over previously reported results. It is interesting to note that in 5 cases checked routinely at 30 days postoperatively the wire had broken but normal position and function were maintained. No wire was used in 5 cases, these being the ones in which only laxity of the ligaments or arthritis alone was found at operation.

Summary

1. Twenty-six cases of acromioclavicular joint injury are reported and discussed.
2. Injury to this joint is seen to cause development of traumatic, degenerative arthritis causing a painful shoulder with limited activity.
3. A satisfactory operative method is presented.
4. It is believed that this method should be offered to all patients except relatively minor ones to prevent painful arthritic acromioclavicular joints which are at least partially disabling.

The opinions or assertions contained in this article are the private ones of the writer and are not to be construed as official or reflecting the views of the Navy Department or the Naval Service at large.

BIBLIOGRAPHY

1. Appel, A. A.: Acromioclavicular Dislocation, Canad. M.A.J. *43*:23-25 (July) 1940.
2. Bohler, L.: The Treatment of Fractures. Vienna, Wilhelm Maudrich, p. 69, 1929.
3. Bowers, R. F.: Complete Acromioclavicular Separation, Diagnosis and Operative Treatment, J. Bone & Joint Surg. *17*:1005, 1935.
4. Broglio, R.: Sulla lussazione dell 'estremita acromiale della clavicola, Gior. Med. Dell' Alto Adega *3*:237, 1931.
5. Budinger, K.: Zur blutigen Behandlung der Luxatio claviculae acromialis und der Bruche des Nasenbeines. Wiener klin. Wchnschr. *13*:595, 1900.
6. Bunnell, S.: Fascial Graft for Dislocation of Acromioclavicular Joint, Surg., Gynec. & Obst. *46*:563, 1928.
7. Cadenat, F. M.: The Treatment of Dislocations and Fractures of the Outer End of the Clavicle, Internat. Clin. *27*:145-169, 1917.
8. Caldwell, G. D.: Treatment of Complete Permanent Acromioclavicular Dislocation by Surgical Arthrodesis, J. Bone & Joint Surg. *25*:368-374 (April) 1943.
9. Carrell, W. B.: Dislocation at Outer End of Clavicle, J. Bone & Joint Surg. *10*:314, 1928.
10. Campbell, W., and Lewis, D.: Practice of Surgery, Vol. II, chap. 5, p. 52.
11. Codman, E. A.: The Shoulder, p. 307, Boston, 1934, Thomas Todd Co.
12. Cooper, E. S.: New Method of Treating Long-Standing Dislocations of the Scapulo-Clavicular Articulation, Am. J. M. Sc. *41*:389, 1861.
13. Copher, G. H.: A Method of Treatment of Upward Displacement of the Acromial End of Clavicle, Am. J. Surg. *22*:507, 1933.
14. Cotton, J. F.: Acromioclavicular Dislocation and Its Repair, New England J. Med. *210*:1025, 1934.
15. Currie, D. I.: An Apparatus for Dislocation of the Acromial End of the Clavicle, British M. J. *1*:570, 1924.
16. Delbet, P. L. E.: Methode de traitement des fractures, avec la collaboration de Marchack (Jambe) et Masse et Lamare (bras). Paris, F. Alcan, 1916.
17. Dunlap, J.: Dislocations of Outer End of Clavicle, California & West Med. *26*:38, 1927.
18. Eickenbary, C. F., and LeCocq, J. F.: The Operative Treatment of Acromioclavicular Dislocations, S. Clin. North America *13*:1305, 1933.
19. Eliason, E. L. in Nelson: Loose-Leaf Surgery, Vol. III, pp. 196-200.
20. Felsenreich, F.: Extraartkulare Fellelung der totalen akromioklavicularen Luxation, Zentralbl. f. Chir. *62*:871, 1935.
21. Furst, A.: Zur operativen Behandlung der Luxatio acromioclavicularis, Zentralbl. f. Chir. *60*:12, 1933.
22. Gray, H.: Anatomy of the Human Body, 21st ed., p. 316, London, Warren H. Lewis.
23. Haggart, G. E.: The Treatment of Acromioclavicular Joint Dislocation, S. Clin. North America, *13*:683-688 (June) 1933.
24. Hamant, A., and Chalnot, P.: Traitement des luxations acromioclaviculaires, Rev. Med. de l'Este, *61*:42, 1933.
25. Hanke, H.: Zur operati ven Behandlung vollstandiger Schlusselbeinverrenkungen, Chirurg *11*:624, 1939.
26. Henry, M. O.: Acromioclavicular Dislocations, Minnesota Med. *12*:431, 1929.
27. Henry, A. K.: Exposures of Long Bones and Other Surgical Methods, New York, Wood, 1927.
28. Howard, N. J.: Acrominoclavicular and Sternoclavicular Joint Injuries, Am. J. Surg. *46*:284-291, 1939.
29. Jones, Sir R.: Injuries to Joints, 2nd ed., London, 1922, Henry Frowde and Hodder and Stoughton, p. 56.

30. King, J. M., Jr., and Holmes, G. W.: Review of 480 Roentgen-Ray Examinations of the Shoulder, Am. J. Roentgenol. *17*:214-218, 1927.
31. Kleinschmidt, O.: Die Ambulante Behandlung der Luxationen, Deutsche Med. Wchnschr. *57*:1503, 1931.
32. Krenn, L.: Zur konservativen Therapie der Luxation im Acromioclaviculargelenk, Chirurg *8*:97, 1936.
33. Lambotte, A.: Chirurgie Operatoire des Fractures, Paris, Masson & Cie, 1913.
34. Lasher, W. W.: Cartilage Injuries. A Clinical Study, Am. J. Surg. *6*:493, 1929.
35. Liberson, F.: The Role of the Coracoclavicular Ligaments in Affections of the Shoulder Girdle, Am. J. Surg. *44*:145-157, 1939.
36. Macey, H. B.: Separation of Acromioclavicular Joint, Proc. Staff Meet., Mayo Clinic, *43*:683, 1936.
37. Meyerding, H. W.: The Treatment of Acromioclavicular Dislocation, S. Clin. North America *17*:1199, 1937.
38. Moffat, B. W.: Separation of the Acromioclavicular Joint, Surg. Gynec. & Obst. *41*:73, 1925.
39. Mollier, S.: Veber die Statik und Mechanik des Menschlichen Schulter gurtels unter normalen und pathologischen Verhaltnisse. Festschrift zum Siebenzigsten Geburtstag von Carl von Kupffer. Venc 1922, Gustav Fischer.
40. Mumford, E. B.: Acromioclavicular Dislocation. A New Operative Treatment, J. Bone & Joint Surg. *23*:799 (Oct.) 1941.
41. Paci, A.: La sutura metallica nella lussazione sopra-acromiale della clavicola, Sperimentale *64*:600, 1889.
42. Poirier, P., and Rieffel, H.: Mechanisme des luxations sus—acromiales des la clavicule, leur treatment par la suture osseuse, Arch. gen. de med. *1*:396-422, 1891.
43. Roberts, S. M.: Acromioclavicular Dislocation; Anatomical Exposure of Outer End of Clavicle and Coracoid Process, Am. J. Surg. *23*:322, 1934.
44. Rost, F.: Die Behandlung der Luxatioacromialis claviculae, Deutsche Med. Wchnschr. *59*:608, 1933.
45. Schneider, C. C.: Acromioclavicular Dislocation. Autoplastic Reconstruction, J. Bone & Joint Surg. *15*:957, 1933.
46. Shaar, C. M.: Upward Dislocation of Acromial End of Clavicle, J. A. M. A. *92*:2083, 1929.
47. Speed, K.: A Text-book of Fractures and Dislocations, Ed. 2, Philadelphia, Lea & Febiger, 1928, p. 242.
48. Thorndike, A., Jr., and Quigley, T. B.: Injuries to the Acromioclavicular Joint, Am. J. Surg. *55*:250-261 (Feb.) 1942.
49. Trynin, A. H.: The Conservative Treatment of Incomplete Dislocation of the Acromioclavicular Joint, J. Bone & Joint Surg. *14*:421-424, 1932.
50. Urist, M. R.: Complete Dislocation of Acromioclavicular Joint, J. Bone & Joint Surg. *28*:813-837 (Oct.) 1946.
51. Watkins, J. T.: An Operation for the Relief of Acromioclavicular Luxations, J. Bone & Joint Surg. *7*:790, 1925.
52. Watson-Jones, R.: Fractures and Other Bone and Joint Injuries, Ed. 2, Baltimore, Williams and Wilkins Co., 1941, p. 307.
53. Wilson, P. D., and Cochrane, W. A.: Fractures and Dislocations, Philadelphia, 1925, J. B. Lippincott Co., pp. 74-75.

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MALIGNANCIES OF THE DUODENUM

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THE duodenum and other small bowel is the one structure in which a tumor is practically always malignant.[58] Even in the pancreas a tumor stands a 20 per cent chance of being benign.[57] Even so, malignancies of the duodenum are rare. Hoffman and Pack[43] say that carcinoma occurs in 0.033 per cent of cases, and that sarcoma has about the same frequency. That is, malignancy may arise in the duodenum about once in 1500 cases. The case being reported is the only proved case on record in the Mississippi Baptist Hospital.

Hamberger is credited with reporting the first duodenal carcinoma in 1746;[43] Alexander the first sarcoma in 1877.[72] Dimetresso reported an epithelioma in 1938.[26]

The origin of duodenal malignancies is, of course, obscure. After careful study Hinton expressed the opinion that ulcers do not contribute, but warns that malignancy may be misdiagnosed as ulcer.[42]

Duodenal malignancies may be about any type of carcinoma, sarcoma, or the less frequently seen varieties of malignancy.

The symptomatology of duodenal malignancies is three times as variable as the pathology. This is true because the duodenum may be divided into three parts, from both the anatomic and the physiologic viewpoints: 1. Supra-ampullary; 2. Ampullary; 3. Infra-ampullary. The localizing late symptom complex depends on the bile and pancreatic juices. In the supra-ampullary lesions bile tends to disappear from the vomitus but not from the stool. In the ampullary region the bile tends to disappear from the intestinal tract. In the infra-ampullary region bile is present in the vomitus but not in the stool. Emphasizing the variableness of the causative factor is the occasional variability of the symptoms as when a polyp sloughs off and symptoms seem to clear. Jaundice, fever, hematemesis and/or melena may come and go; pain may or may not be present. Even obstruction can temporarily abate.

One ordinarily considers these lesions to be rapidly progressive. However, it is surprising how often the history of these patients is long drawn out as to digestive complaints; particularly constipation, diarrhea, or alternating between the two. Quite often the history suggests the type of patient that you and I relegate to the chronics and put on the shelf with varicolored pills.

X-ray is useful, but too rarely successful in early diagnosis. Due to the anatomy of the region, small defects can easily be overlooked.

Even in the late cases the diagnosis is none too accurate. There are various trick procedures such as simultaneously filling the stomach and colon with barium to aid in identifying large tumors.

Treatment is entirely surgical. In carcinoma x-ray therapy is of little use,[45] but x-ray therapy is useful in sarcomatous lesions.[71] It is interesting to note the advance of treatment although the results are still not satisfactory. In the series of 134 proved cases from 1907 through 1946 appended, palliative surgery began in 1915; segmental resection in 1924; and radical extirpation in 1938.

There are 97 carcinomas: 61 males, 34 females, 2 not stated.

There are 36 sarcomas: 20 males, 14 females, 2 not stated.

The youngest carcinoma patient was 18; the youngest sarcoma 6.

Of the 134 cases, 41 were of autopsy only. There are 93 operations which tabulate as follows:

21 exploratory laparotomies with a hospital mortality of 71 per cent

28 palliative operations with a hospital mortality of 55 per cent

32 segmental resections with a hospital mortality of 22 per cent

7 one stage resections with a hospital mortality of 14 per cent

5 two stage resections with a hospital mortality of 40 per cent

There are 3 five year cures listed:

Case 15—Segmental resection of an infra-ampullary sarcoma.
Case 116—Segmental resection of a supra-ampullary carcinoma.
Case 130—One stage resection with pancreatic anastomosis for a periampullary carcinoma.

Eusterman in 1925 reported 15 cases treated at the Mayo Clinic by palliative surgery,[30] and Dixon in 1946 reported the whole Clinic series of 49 cases with 4 segmental and 4 radical resections.[27] Only one case of the latter report is included in this discussion.

The allied conditions of biliary duct, pancreatic duct, common duct, pancreatic malignancies and tumors are far more frequent than duodenal malignancies. They may be so closely similar as to require serial microscopic study for accurate differentiation. In these allied conditions, Halsted is credited with the first removal of the ampulla of Vater in 1898;[47] Codivillar with performing "A block removal of the head of the pancreas with the duodenum in one stage in 1898, completing the operation by a Roux Y type gastrointestinal restoration and cholecystojejunostomy."[57] Whipple did the first successful one stage resection in 1940.[95] It is he[95-7] who has fathered duodeno-pancreatic resection by his finding that pan-

creatic secretion is not necessary to life in the human. In 1935 he published a two stage procedure which became the standard where the pancreas was to be oversewn. He later altered his technic from cholecystogastrostomy to cholecystojejunostomy in Y because of the frequency of hepatitis in the first procedure, and from the two to a one stage operation because he considered the mortality rate more favorable when used in conjunction with the more recent improvements in specific and general surgical care. Orr[67] belongs to the school advocating pancreatic anastomosis. He has set up a list of requirements for any duodeno-pancreatic resection procedure which is worth repeating: 1. Biopsy. 2. Removal of sufficient tissue. 3. Use of the common duct for anastomosis. 4. Location of gall duct anastomosis proximal to the stomach. 5. Anastomosis of the pancreas and jejunum. Strode's technic[83] meets these criteria: After removal of the duodenal horseshoe at the ligament of Treitz, the jejunal end is anastomosed to the pancreatic stump. A loop is then brought up and anastomosed to the common duct, then on the way down to the stomach as in a Pollya resection. This sounds simple, and is, when compared to some of the complicated procedures found. The argument about the need for pancreatic juice is still unsettled; for some reason one human will have normal absorption and stools with absence of pancreatic secretion in the intestines proved by subsequent autopsy, which also revealed a normal liver,[96] while another will have defective absorption and intestinal disorders.

Case Report

W. H. M., a white male, aged 50, was complaining of nausea, vomiting, and jaundice when referred to me on Nov. 8, 1945.

Present Illness: The nausea had begun about 4 months previously and had been extreme. He stated that in September only drastic cathartics gave him any relief from constipation. He had lost 17 pounds. Vomiting had gradually become more progressive to where little food was retained. The jaundice was first noted 25 days before. It and the attendant pruritus had made hypodermics necessary for relief, but he had had no pain.

Past History: In 1918 he had had a bilateral hernial repair.

He had been treated for colon trouble and allergy (but not asthma) for several years. Complete x-ray study on July 26, 1945, revealed no abnormality.

Family History: The patient's father had died of a cancer.

Physical: Examination showed a well developed and still well nourished white man about 50 years old who was deeply jaundiced and constantly scratching. He has a habit of rolling his eyes up with his lids open.

Teeth good. Tonsils present.

Heart and lungs normal. Blood pressure 120/80.

Abdomen: No distention is present. There is moderate tenderness over the epigastrium but no mass is made out. Bilateral hernial scars are noted.

Laboratory: No helpful findings.

X-ray of gallbladder area shows no stone.

On November 8, after glucose drips and vitamin K therapy, he was operated on, the preoperative diagnosis being obstructive jaundice.

Operation: Under ether anesthesia the abdomen was opened through an upper right rectus incision. The gallbladder was distended but no stones were palpated. The common duct was dilated but no stone was palpated. The pancreas was palpated. The head felt hard but was smooth. The abnormal enlargement was the size of a lemon and seemed strictly limited to the head of the organ. It was fully movable, not attached to the stomach or colon. There was apparently no attachment other than the normal pancreatic attachments. Even in the duodenal horseshoe, which was stretched tight with resultant decrease of luminal size, there was no apparent attachment except to the duodenum. No metastasis was palpable. There was no abnormal fluid in the abdomen.

Operative procedure: An isoperistaltic anterior gastrojejunostomy was done paralleling the lower border of the stomach proximal to its middle. A cholecystojejunostomy was done. On the level of the stomach between the loops extending to the gallbladder, an entero-anastomosis was done.

The postoperative course was uneventful. He was discharged from the hospital in about 2 weeks. On November 23 in the office his weight was 146. His appetite was good, he had no nausea, and was free of jaundice.

On December 15 x-rays were taken which showed the gastroenterostomy stoma to be functioning.

After a pleasant Christmas, with all the trimmings, he reported for examination on December 31. An x-ray of the chest was normal. The matter of a second operation was freely discussed and he decided to take the chance.

On Jan. 8, 1946, under ether anesthesia the second operation was done. The gastroenterostomy stoma was checked and found adequate. The tumor felt harder but slightly less large. It felt movable.

Operative procedure: The stomach was divided distal to the gastrojejunostomy, the proximal stump being repaired as a gastrectomy. The distal end was closed with a temporary suture. The gastrohepatic omentum was divided to the right. The peritoneum to the right of the duodenum was divided and the duodenum rotated to the left and downward. The back side of the pancreatic head presented a cyst which distorted the anatomy. The tumor seemed to end before the superior mesenteric artery was reached. The duodenum was then divided proximal to the superior mesenteric artery, the distal end being closed with care, the proximal end closed temporarily. With these structures divided it was possible to rotate the head of the pancreas to the left. It was found to be adherent to the portal vein and superior mesenteric vessels, and when that area was attacked by blunt dissection the patient went into shock. Transfusions brought him back, but every time this area was attacked his condition immediately became grave. A catheter was placed in the distal end of the stomach, a cigarette drain was placed over the distal end of the duodenum, and the abdomen closed around them.

On one occasion some bleeding occurred from the growth and it was extremely difficult to control by suture.

The postoperative course was progressively downward. After about the

first week he vomited practically everything taken. Drainage became extreme and he was kept clean by suction in the wound. A tender mass formed in the left side of the abdomen. He died on Jan. 24, 1946. An autopsy was done.

Gross Description

In the head of the pancreas is a firm slightly nodular, fairly well circumscribed gray mass measuring 60 by 53 by 40 mm. It is readily separated from the pancreatic tissue but is firmly attached to the first and second portion of the duodenum. In the second portion of the duodenum the tumor protrudes into the lumen but is covered by smooth duodenal mucosa. On section the mass is solid, friable and has a gray color. The duct of Wirsung is not dilated. The lobules of yellowish gray pancreas are separated by moderately abundant semi-translucent white tissue. In the peri-pancreatic fat there are yellowish gray opaque foci.

Microscopic Description

Section through the wall of the duodenum includes underlying tumor which has involved the muscular coat. Large areas of the tumor show ischemic necrosis and the contained blood vessels are often thrombosed. Where tumor is well preserved it is composed of spindle-shaped cells arranged in intertwining bundles. The cytoplasm is lightly acidophilic and the nuclei are round, oval or sometimes lobulated and hyperchromatic. Mitotic activity is slight. Capillaries are moderately abundant throughout the tumor. The mucosa of the duodenum overlying the tumor has a normal appearance.

Microscopic Diagnosis

Spindle cell myosarcoma of duodenum.

In concluding allow me to quote Whipple: "Many more cases with five year survivals will be required before valid claims can be made for the operation as done at present. But it must be remembered that these patients untreated have an average of six months survival from onset of symptoms to death. If only the patients could and would be referred to the surgeon before they are studied to death, while the cancer is smaller and localized and before the patients are seriously ill with weeks of obstructive jaundice, a far lower operative mortality and a much longer survival would result."

BIBLIOGRAPHY

1. Aguirre, J. A.; Senorans, A. J., and Mazzini, E. J. B.: Cancer primitivo de duodeno, Actual. med. mund. *13*:95-116 (May) 1943.
2. Anderson, D. H., and Doob, E. F.: Leiomyosarcoma of the Duodenum, Arch. Path. *14*:795-802 (Dec.) 1933.
3. Archibald, J. S.: Carcinoma of the Duodenum, Report of Case, Illinois M. J. *58*:62-65 (Jan.) 1930.
4. Arisz, L.: Duodenal Ulcer with Carcinoma, Acta Radiol. *13*:41-42, 1932.
5. Bajer, I.: Case of Primary Carcinoma of Duodenum, M. Press *214*:46-48 (July 18) 1945.
6. Bastos, E. S.: Carcinoma primitivo do duodeno, Brazil-med. *49*:734-741 (Aug. 17) 1935.

TABLE I

1	Case number			1	2	3	4
2	Year reported 19—			07			
3	Initials						
4	Age			64	40	39	89
5	Male					x	x
6	Female			x	x		
7	Carcinoma			x	x	x	x
8	Sarcoma						
9	REGION	Supra. Amp.		x			
10		Peri Amp.			x		
11		Infra. Amp.				x	x
12	OPERATION	Exploratory		x			
13		Palliative					
14		RADICAL	Segmental				
15			1st Stage				
16			2nd Stage				
17			Panc. Anast.				
18			Panc. Suture				
19	Hospital Deaths			x			
20	Discharged Alive						
21	Lived (mo.)						
22	Autopsy			x	x	x	x
	AUTHOR			GEISER			
	References			35			

TABLE I (*Continued*)

1	5	6	7	8	9	10	11	12
2				15	17	20	20	21
3				JC		MO		
4	81	68	57	52	43	63	54	6
5	x		x			x	x	x
6		x		x	x			
7	x	x	x		x	x	x	
8				x				x
9			x					
10		x			x		x	
11	x					x		x
12				x				
13					x	x	x	
14								
15								
16								
17								
18								
19				x	x	x	x	
20								
21								
22	x	x	x	x	x	x	x	x
AUTHOR	GEISER			FORGUE	THAYSEN	DEAVER	VERVLOT	MACERA
	35			31	87	24	92	52

TABLE I (*Continued*)

1	13	14	15	16	17	18	19	20
2	24	24	25	25	26	27	27	28
3		LPC	IJF					
4	40	64	73	62	78	61	17	59
5	x	x		x				x
6			x		x	x	x	
7		x			x	x	x	x
8	x		x	x				
9								x
10					x			
11	x	x	x			x	x	
12								
13	x	x		x	x		x	
14			x			x		x
15								
16								
17								
18								
19					x			
20		x	x	x		x		x
21			60	24		3		24
22	x	x			x		x	

AUTHOR	GIANI	VICKERS	MILLER	STRAUSS	MASS. GEN. 12063	PAUCHET	MOSTOWSKA	DEWIS
	36	93	60	82	15	69	61	25

TABLE I (*Continued*)

1	21	22	23	24	25	26	27	28
2	29	30	31	31			32	32
3		PHY		AJ	JM	FO		WA
4	47	64	41	36	55	60	36	64
5		x		x	x	x		x
6	x		x				x	
7	x	x		x	x	x		x
8			x				x	
9		x	x					x
10				x				
11	x				x	x	x	
12	x	x					x	x
13						x		
14			x					
15								
16								
17								
18								
19		x				x	x	x
20			x					
21			12					
22	x	x		x	x	x	x	x
AUTHOR	YLVISAKER	ARCHIBALD	PFUNDT	MEYER			LOYER	MANTEER
	102	3	70	62			51	56

TABLE I (*Continued*)

1	29	30	31	32	33	34	35	36
2		32		32				33
3	HS			PHY				
4	71			83	58	73	50	37
5	x	x	x	x		x	x	x
6					x			
7	x	x	x	x	x	x	x	
8								x
9		x	x	x	x		x	
10						x		x
11	x							
12	x				x			
13								
14		x	x					
15								
16								
17								
18								
19	x							
20		x	x		x			
21		36	36		3			
22			x	x	x	x	x	x
AUTHOR	MANTEER	ARISZ		RUTISHAUSER (1929) (1931)				ANDERSEN
	56	4		75				2

TABLE I (*Continued*)

1	37	38	39	40	41	42	43	44
2	34	34	34	34	35	35	35	35
3			CP		JG	KN	NC	AN
4	53	47	45	50	48	59	34	67
5		x	x		x	x	x	x
6	x			x				
7			x	x		x	x	x
8	x				x			
9	x	x	x					x
10				x			x	
11					x	x		
12					x	x		
13		x	x					x
14	x							
15								
16								
17								
18								
19		x	x			x		x
20	x				x			
21					1			
22		x		x	x	x	x	x
AUTHOR	WILLIAMSON	DIMITRESCO	DARDINSKI	LISA	PREY	SALA	STARTZ	BASTOS
	100	26	22	50	73	76	81	6

TABLE I (*Continued*)

1	45	46	47	48	49	50	51	52
2	35	36	37					
3	MOB	JS	MB	DH	BK	CS	ED	FR
4	46	54	48	28	60	47	56	36
5		x						
6	x		x	x	x	x	x	x
7	x		x		x	x	x	x
8		x		x				
9		x	x		x			
10						x	x	x
11	x			x				
12					x			x
13			x	x			x	
14	x							
15								
16								
17								
18								
19	x		x		x		x	
20				x				x
21				6				4
22	x	x	x	x	x	x	x	x

AUTHOR	DAVIS	SEYMOUR	MEMORIAL HOSPITAL	ST. JOHN'S, L. I.	NEW HAVEN
	23	78	43		

TABLE I (*Continued*)

1	53	54	55	56	57	58	59	60
2								
3	CA	HB	AB	SA	JR	WF	GB	AB
4	52	78	71	18	51	45	45	58
5		x	x	x	x	x		x
6	x						x	
7	x	x	x	x	x	x	x	x
8								
9								
10	x		x	x		x	x	
11		x			x			x
12								x
13				x		x	x	
14					x			
15								
16								
17								
18								
19						x		x
20				x	x		x	
21							24	
22	x	x	x					
AUTHOR	N. Y. POLYCLINIC		PATTERSON GEN.	ERDMAN				LENOX HILL
	43							

TABLE I (*Continued*)

1	61	62	63	64	65	66	67	68
2				37				
3	CG	LH	MB	JP	NC	MC	AW	IT
4	45	70	51	35	69	58	85	66
5				x	x	x		
6	x	x	x				x	x
7	x	x	x	x	x	x	x	x
8								
9			x	x	x	x	x	x
10	x							
11		x						
12				x				
13								
14								
15								
16								
17								
18								
19				x				
20								
21								
22	x	x	x	x	x	x	x	x
AUTHOR	LENOX HILL			STEWART				
	43			84				

TABLE I (*Continued*)

1	69	70	71	72	73	74	75	76
2		37	37	37	37	37	37	
3	EH					HM	JP	MD
4	44	64	39	42	56	33	33	68
5	x	x	x		x	x	x	
6				x				x
7	x	x	x			x	x	
8				x	x			x
9	x	x	x	x	x		x	x
10								
11						x		
12						x		
13								
14				x	x		x	x
15								
16								
17								
18								
19				x	x	x		
20							x	x
21							3	6
22	x	x	x	x	x	x		
AUTHOR	STEWART	WOODS	JARRELL	MASS. GEN. 23261	MASS GEN. 23262	HOWES	HOLUBEC	
	84	101	48	15	15	46	44	

TABLE I (*Continued*)

1	77	78	79	80	81	82	83	84
2	38	38	38	38	38	38	38	38
3			VT	LN				JT
4	54	54	49	47	66	34	47	69
5	x		x		x	x	x	x
6		x		x				
7	x	x		x		x	x	x
8			x		x			
9	x					x		
10			x		x			x
11		x		x			x	
12								
13	x			x	x			
14		x	x					
15								
16						x	x	
17								
18						x	x	
19	x				x	x	x	
20		x	x					
21		12	8					
22	x				x	x		x
AUTHOR	NICOLINI	HANDFIELD-JONES	MENDILLO	POLLOCK	BOSSAERT	MASS. GEN. 24162	CLAIBORNE	DUCKWORTH
	64	37	59	71	10	16	20	28

TABLE I (*Continued*)

1	85	86	87	88	89	90	91	92
2	39	39		39	39		39	39
3								
4	54	48	40	54	60	64	73	50
5	x	x	x			x		
6				x	x		x	x
7	x			x	x	x	x	
8		x	x					x
9	x			x			x	
10								
11		x	x		x	x		x
12								
13		x	x					x
14	x			x	x			
15								
16								
17								
18								
19				x				
20	x	x	x		x			
21	7	16	50		30			
22		x		x		x	x	
AUTHOR	WIIG	PRESENT 428-607 464-534		MASCIOTTRA	BERGENDAL	BERGENDAL	GASBARRINI	TASCA
	98	72		55	7		34	85

TABLE I (*Continued*)

1	93	94	95	96	97	98	99	100
2	39	40	40	40	40			
3	PV							
4	63	48	38	42	29	7	53	60
5	x			x	x	x	x	x
6		x	x					
7		x	x					
8	x			x	x	x	x	x
9		x		x				
10					x	x	x	
11	x		x					x
12							x	x
13								
14	x		x	x				
15								
16								
17								
18								
19							x	x
20	x		x	x				
21			5					
22	x	x			x	x	x	x

AUTHOR	FOSHEE	RAMOND	HARRINGTON	ITIKAWA	MARKOVIC
	32	74	40	48	54

TABLE I (*Continued*)

1	101	102	103	104	105	106	107	108
2	41		41	41	41	41	41	41
3								
4	43	52	43	66	51	52	42	65
5		x		x	x			
6	x		x			x	x	x
7		x	x	x	x	x		
8	x						x	x
9				x	x			x
10	x	x	x					
11						x	x	
12							x	
13						x		
14					x			x
15	x	x						
16			x					
17			x					
18	x	x						
19		x			x	x	x	x
20	x		x					
21	1		1					
22		x		x	x	x	x	x
AUTHOR	CHIASSERINI		HUNT 479-44	PALAZUELOS	BONORINO	MASS. GEN. 27092	MCCULLOUGH	HENNING
	18		47	68	9	17	63	41

TABLE I (*Continued*)

1	109	110	111	112	113	114	115	116
2	41	42	42	42	43	43	43	43
3						PO		ED
4	59	45	45	46	42	67	41	27
5	x	x	x	x		x	x	x
6					x			
7	x		x	x		x	x	x
8		x			x			
9		x		x		x		x
10			x		x			
11	x						x	
12								
13	x		x				x	
14		x		x				x
15					x			
16								
17								
18					x			
19	x							
20		x	x	x	x		x	x
21		2	27	24	2			60
22					x	x	x	
AUTHOR	CRESSY	GARCIA	TREMOR	VEAL	BISGARD	MANTEL	AGUIRRE	BURKE
	21	33	89	91	8	53	1	13

TABLE I (*Continued*)

1	117	118	119	120	121	122	123	124	125
2	43	43			44	44	44	45	45
3	JB				JMCH		IH		BR
4	57	63	80	48	63	67	30	44	66
5	x	x	x	x	x	x		x	x
6							x		
7	x	x	x	x	x	x			x
8							x	x	
9		x		x		x			
10	x							x	x
11			x		x		x		
12				x			x		
13									
14					x				
15	x							x	x
16									
17	x								
18								x	x
19									
20	x			x	x			x	x
21	14				9				5
22		x	x	x		x	x		

AUTHOR	CHILD	HOWARD	SHALLOW	THORSTAD	WILLIAMS	STRODE	ORR
	19	45	80	88	99	83	66

TABLE I (*Continued*)

1	126	127	128	129	130	131	132	133	134
2	45	45	46	46	46				
3		WHM		ER	FK		GJ	GM	CT
4	25	50	52	33	52				
5	x	x			x				
6			x	x					
7	x		x		x	x			x
8		x		x			x	x	
9						x			
10	x	x	x		x			x	
11				x			x		x
12	x								
13									
14				x		x	x	x	x
15					x				
16		x	x						
17					x				
18			x						
19	x	x						x	
20		x	x	x	x	x	x		x
21			32	6	94	12	36		3
22		x							

AUTHOR	BAJER	AUTHOR'S CASE	DIXON	EMMETT	BRUNSCHWIG
	5		27	29	12

7. Bergendal, A.: Contribution to the Knowledge of Primary Duodenal Cancer, Acta Radiol. *20*:417-426, 1939.
8. Bisgard, J. D., and Cochran, R. M.: Primary Sarcoma of Duodenum, Am. J. Surg. *61*:425-429 (Sept.) 1943.
9. Bonorino Udaondo, C.: Cancer primitivo del duodeno, Prensa med. argent. *28*:1018-1020 (May 7) 1941.
10. Bossaert, P.: Un cas de lymphosarcome du duodenum; Considerations sur les tumeurs duodenales, J. de chir. et ann. Soc. belge de chir. *37-35*:182-189 (June) 1938.
11. Brunschwig, A.: One Stage Pancreatoduodenectomy, Surg., Gynec. & Obst. *77*:581-584 (Dec.) 1943.
12. Brunschwig, A., and Tiholiz, I. C.: Surgical Treatment of Malignant Tumors of the Duodenum Exclusive of Those Arising from the Papilla of Vater, S. Clin. North America *26*:163-175 (Feb.) 1946.
13. Burke, E.; Perkel, L. L., and Gnassi, A. M.: Duodenal Carcinoma, Am. J. Surg. *62*:267-271 (Nov.) 1943.
14. Cabot case 12063: Adenocarcinoma of the Duodenum, Boston M. & S. J. *114*:267-270, 1926.
15. Cabot case 23261: Neurogenic Fibrosarcoma of the Duodenum, New England J. Med. *217*:27-29 (July 1) 1937.
16. Cabot case 24162: Carcinoma of the Duodenum, New England J. Med. *218*:687-689 (April 21) 1938.
17. Cabot case 27092: Carcinoma of the Duodenum, New England J. Med. *224*:386-388 (Feb. 27) 1941.
18. Chiasserini, A.: Interventi radicali per tumeri del duodeno, Policlinico (sez. prat.) *48*:649-659 (April 14) 1941.
19. Child, C. G.: Carcinoma of the Duodenum, Ann. Surg. *118*:838-842 (Nov.) 1943.
20. Claiborn, L. N., and Dobbs, W. H. G.: Carcinoma of Third Segment of Duodenum; Report of Cases, Surgery *4*:97-102 (July) 1938.
21. Cressy, N. L.: Carcinoma of the Duodenum, Connecticut M. J. *5*:818-820, 1941.
22. Dardinski, V. J.: Primary Carcinoma of the Duodenum, Am. J. Path. *10*:313-317, 1934.
23. Davis, C. R.: Carcinoma of the Duodenum; Case Report, Am. J. Cancer *23*:337-338 (Feb.) 1935.
24. Deaver, J. B., and Ravdin, I. S.: Carcinoma of the Duodenum, Am. J. M. Sc. *159*:469, 1920.
25. Dewis, J. W., and Morse, G. W.: Primary Adenocarcinoma of the Duodenum, New England J. Med. *198*:383-401, 1928.
26. Dimitresco-Popovici: Le cancer du duodenum, Presse Med. *42*:1978-1980, 1934.
27. Dixon, C. F. et al: Malignant Lesions of the Duodenum, Surg., Gynec. & Obst. *83*:83-93 (July) 1946.
28. Duckworth, W. D.: Primary Cancer of the Duodenum; Report of Case, Am. J. Roentgenol. *40*:377-380, 1938.
29. Emmett, J. M., and Dreyfuss, M. L.: Malignant Tumors of the Small Bowel, Ann. Surg. *123*:859-965 (May) 1946.
30. Eusterman, G. B.; Berkman, D. M., and Swan, T. S.: Primary Carcinoma of the Duodenum, Ann. Surg. *82*:153-163, 1925.
31. Forgue, E., and Chauvin, E.: Le cancer primitif et intrinsique (non Vaterien) du Duodenum, Rev. de chir., 1914-15, *34*:470-582 (Dec.) 1915.
32. Foshee, J. C., and McBride, W. R. L.: Leiomyosarcoma of the Duodenum; Report of a Case and Review of the Literature, J. A. M. A. *112*:2497-2500 (June 17) 1939.
33. Garcia Baron, A.: Sarcoma primario de la 1.a y 2.a porcion del duodeno. Reseccion con curacion inmediata, Rev. clin, espan. *6*:182-185 (Aug. 15) 1942.
34. Gasbarrini, A.; Corazza, G., and Motolese, G.: Adenocarcinoma primitivo del duodeno, Policlinico (sez.prat.) *46*:1787-1794 (Oct. 9) 1939.
35. Geiser, J. F.: Uber Duodenalkrebs. Klinisch-pathologisch-anatomischer Beitrag, Deut. Ztschr. f. Chir. *86*:41-107, 1907.

36. Giani, E.: Sarcoma primitivo del duodeno (considerazioni anatomopathologiche e cliniche), Arch. di patol. *3*:9-25, 1924.
37. Handfield-Jones, R. M.: Intrinsic Carcinoma of the Duodenum; with a Report of Successful Removal, Lancet *2*:1168-1170, 1938.
38. Hansen, E. H.: Cancer of the Duodenum, Primary and Secondary, Acta chir. Scandinav. *80*:295-316, 1938.
39. Harbin, W. P.; Harbin, W. P., Jr., and Harbin, L.: Primary Carcinoma of the Duodenum, Surgery *10*:961-965, 1935.
40. Harrington, S. W., and Ganshorn, J. A.: Leiomyosarcoma of the Third Portion of the Duodenum; Report of Case, Proc. Staff Meet., Mayo Clin. *15*:77 (Jan. 31) 1940.
41. Henning, B. H., and Garland, L. H.: Leiomyosarcoma of the Duodenum, Radiology *37*:353-357, 1941.
42. Hinton, J. W.: Does Carcinoma of the Duodenum Ever Arise from Duodenal Ulcers?, Am. J. M. Sc. *181*:843-850, 1931.
43. Hoffman, W. J., and Pack, G. T.: Cancer of the Duodenum, Arch. Surg. *35*:11-62 (July) 1937.
44. Holubec, K.: DVA pripady malignich nadoru duodena, Casop. lek. cesk. *76*:1586-1587 (Sept. 10) 1937.
45. Howard, J. W.: Carcinoma of the Duodenum, Am. J. M. Sc. *206*:735-746 (Dec.) 1943.
46. Howes, W. E.: The Duodenum; Roentgenologically Considered and Including One Case of Primary Carcinoma, New York State J. Med. *37*:1997-2005, 1937.
47. Hunt, V. C.: Surgical Management of Carcinoma of the Ampulla of Vater and of the Periampullary Portion of the Duodenum, Ann. Surg. *114*:570 (Oct.) 1941.
48. Itikawa, H.: Drei Falle von Leiomyom des Verdauungstraktes. 11. Abteillung. Leiomyosarcoma duodeni, Arch. f. jap. Chir. *17*:1270-1271, 1940.
49. Jerrell, P. M.: Huntington's Chorea, Adenocarcinoma of Duodenum, Chronic Encephalitis, M. Bull. Vet. Admin. *13*:269-272 (Jan.) 1937.
50. Lisa, J. R.; Levine, J., and Fitzhugh, W. M.: Primary Carcinoma of the Duodenum, J. Lab. & Clin. Med. *20*:150-154, 1934.
51. Loyer; Belot, and Espieux: Sarcome lymphoblastique de l'ovarie et du mesentere a point de depart intestinal, Ann. d'anat. path. *9*:1018-1021, 1932.
52. Macera, J. M.: Consideraciones clinicas sobre una linfosarcomatosis de duodeno en la infancia, Arch. latino. am. de pediat. *15*:206-212, 1921.
53. Mantell, F. J., and Bradford, W. H.: Primary Carcinoma of the Duodenum, M. Bull. Vet. Admin. *20*:108-109 (July) 1943.
54. Markovia-Peicic, B.: O limfosarkomu duodenuma, Lijecn. vjes., Zagreb *62*:149-152 (March) 1940.
55. Masciottra, R. L.: Carcinoma primitivo del bulbo duodenal, Rev. Asoc. med. argent. *53*:820-821, 1939.
56. Mateer, J. G., and Hartman, F. W.: Primary Carcinoma of the Duodenum; Clinical and Pathologic Aspects, with Differential Diagnosis, J. A. M. A. *94*:1853-1859, 1932.
57. Maxeiner, S. R.: Carcinoma of Ampulla of Vater, Minnesota Med. *28*:225-227 (March) 1945.
58. Medinger, F. G.: Malignant Tumors of the Small Intestine, Surg., Gynec. & Obst. *69*:299-305, 1939.
59. Mandillo, A. J., and Koufman, W. B.: Diverticulum and Sarcoma of the Duodenum, New England J. Med. *219*:432-433, 1938.
60. Miller, C. J.: Sarcoma of the Duodenum, New Orleans M. & S. J. *78*:27-28, 1925.
61. Mostowska, Z.: Okrago-komorskowy miesak swunastnicy, Polska gaz. lek. *6*:250-251, 1927.
62. Meyer, J., and Rosenberg, D. H.: Primary Carcinoma of the Duodenum; Report of 4 Cases with a Review of the Literature, Arch. Int. Med. *48*:917-941, 1931.
63. McCullough, K.: Leiomyosarcoma of the Duodenum, New York State J. Med. *44*:1248-1249 (June 1) 1944.
64. Nicolini, R. C.: Cancer primitovo del duodeno, Bol. y trab. de la Soc. de cir. de Buenos Aires *22*:666-680, 1938.

65. Orr, T. G.: Resection of Duodenum and Head of Pancreas for Carcinoma of the Ampulla, Surg., Gynec. & Obst. *73*:240-243 (Aug.) 1941.

66. Orr, T. G., and Walker, G. A.: Pancreaticoduodenectomy for Primary Carcinoma of the Duodenum, Surg., Gynec. & Obst. *80*:149-151 (Feb.) 1945.

67. Orr, T. G.: Pancreaticoduodenectomy for Carcinoma of the Ampulla and Ampullary Region, Surgery *18*:144-158 (Aug.) 1945.

68. Palazuelos, G. P., and Lerner, M. J.: Sindroma pilorico de etiologia rara: Carcinoma primitivo del duodeno, Rev. med. de Chile *69*:270-273 (May) 1941.

69. Pauchet, Victor, and Luquet: Cancer de la quartrieme portion du duodenum, Bull. Acad. de med., Paris *97*:276-279, 1927.

70. Pfundt, W.: Uber ein Spindelzellensarkom des Duodenums, Arch. f. klin. Chir. *163*:488-498, 1931.

71. Pollack, S.: Primary Carcinoma of the Third Portion of the Duodenum, Radiology *41*:362-363, 1938.

72. Present, A. J.: Primary Lymphosarcoma of the Duodenum, Am. J. Roentgenol. *41*:545-548, 1939.

73. Prey, D.; Foster, J. M., Jr., and Dennis, W.: Primary Sarcoma of the Duodenum; Report of a Case, Arch. Surg. *30*:673-684, 1935.

74. Ramond, F.: Cancer Primitif du duodenum, Arch. d. mal. de l'app. digestif, *29*:1075-1076, 1940.

75. Rutishauser, E.: Zur Pathologie des Duodenalkrebses, Frankfurt. Ztschr. f. Path. *43*:133-166, 1932.

76. Sala, A. M.: Pathological Rarities in Cancer, Radiology *25*:437-439, 1935.

77. Von Salis, H. W.: Case of Sarcoma of the Duodenum, Deutsche Ztschr. f. Chir. *150*:180; abst. J.A.M.A. *76*:972, 1921.

78. Seymour, W. J., and Gould, S. E.: Leiomyosarcoma of the Duodenum, Am. J. Cancer *28*:572-578, 1936.

79. Shackelford, R. T.; Fisher, A. M., and Firor, W. B.: Duodenal Tumor of Unusual Character, Ann. Surg. *116*:864, (Dec.) 1942.

80. Shallow, T. A. et al: Primary Carcinoma of Third Portion of Duodenum, Surgery *16*:939-946 (Dec.) 1944.

81. Startz, I. S.: Duodenal Carcinoma, Radiology *25*:688-697, 1935.

82. Strauss, A. A. et al: Sarcoma of the Duodenum and Stomach, S. Clin. North America *5*:977-984, 1925.

83. Strode, J. E.: Radical Duodenopancreatectomy, Surgery *18*:115-129 (July) 1945.

84. Stewart, H. L., and Lieber, M. M.: Carcinoma of the Suprapapillary Portion of the Duodenum, Arch. Surg. *35*:99-129, 1937.

85. Sutherland, C. G.: The Pancreas, Am. J. M. Sc. *204*:769-779 (Nov.) 1942.

86. Tasca, M.: Linfosarcoma primitivo del duodeno. Studio radiologico, Boll. e. men, Soc. piemontese di chir. *8*:264-269, 1939.

87. Thaysen, T. E. H.: Duodenalkarcinom med intermitterende Stenosetilfaelde, Ugesk. f. Laeger *79*:1559-1561, 1917.

88. Thorstad, M. M. et al: Primary Carcinoma of Duodenum, Harper Hosp. Bull *2*:12 (Jan.) 1944.

89. Tremor, V. F.: Papillary Carcinoma of the Duodenum, M. Bull. Vet. Admin. *18*:313 (Jan.) 1942.

90. Vaughn, A. M.: Primary Carcinoma of the Ampulla of Vater, Am. J. Surg. *52*:489-493 (June) 1941.

91. Veal, J. R.; Weinstein, J. J., and Conneen, L. W.: Adenocarcinoma Arising in a Papilloma of the First Portion of the Duodenum, M. Ann. District of Columbia *11*:59-61 (March) 1942.

92. Vervloet, C. G.: Primair carcinoma duodeni, Nederl, Tijdschr. v. Geneesk. *1*:181-182, 1920.

93. Vickers, D. M.: Carcinoma of the Duodenum, Ann. Surg. *79*:239-243, 1924.

94. Waugh, J. M.: Resection of the Head of the Pancreas and Duodenum; Operative Technic, S. Clin. North America *26*:941-948 (Aug.) 1946.

95. Whipple, A. O.; Parsons, W. B., and Mullins, C. R.: Treatment of Carcinoma of the Ampulla of Vater, Ann. Surg. *102*:763, 1935.

96. Whipple, A. O., and Banman, L.: Observations on the Pathologic Physiology of the Insular Anterior External Secretory Functions of the Human Pancreas, Am. J. Sc. *102*:21-36, 1941.

97. Whipple, A. O.: Present Day Surgery of the Pancreas, New England J. Med. *226*:515-526 (March 26) 1942.

98. Wiig, N.: Et tilfelle av primaer kancer i bulbus duodeni, Nord. Med. (Med. rev., Bergen) *2*:1767-1769, 1939.

99. Williams, M. H. L.: A Case of Perforating Leiomyosarcoma of the Duodenum Associated with Parturition, M. J. Australia *1*:585-586, 1933.

100. Williamson, C. S.: Sarcoma of the Duodenum Treated by Partial Duodenectomy, West. J. Surg. *42*:207-210, 1934.

101. Woods, J. O.: Primary Carcinoma of the Duodenum, Pennsylvania M. J. *41*:27-31, 1937.

102. Ylvisaker, R. S.: Carcinoma of the Third Portion of the Duodenum, with a Brief Review of the Literature, Minnesota Med. *12*:351-354, 1929.

MEIGS' SYNDROME

Report of a Case of Fibroma of the Ovary with Ascites

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THAT there is a clear-cut syndrome consisting of the collection of colloidal fluid in the peritoneal and pleural cavities associated with fibroma of the ovary is well established by evidence of numerous reports in the literature following Meigs' description of it in 1937.[1-9] The condition was first described by Cullingworth in 1879,[10] who enumerated the salient features of the condition, stating the clinical picture resembles malignancy of advanced degree with pelvic tumor, ascites, and pleural effusion, though actually only a fibroma is present in the ovary. Tait[11] emphasized the fact that ascites and pleural effusion associated with abdominal tumors does not always imply malignancy and reported a case in 1892. Mapes[12] described a case of ascites with fibroma of the ovary in 1909. Kelly and Cullen[13] reported a case of leiomyomata of the uterus with ascites and hydrothorax, with recovery following hysterectomy. Titus[14] in 1913 reported a case with ascites but did not mention hydrothorax. Caro[15] reported a case having both hydrothorax and ascites in association with a large fibroma of the ovary. Owen[18] in 1923 reported a case of fibroma of the ovary with ascites and hydrothorax. Hoon[19] reviewed 55 cases of fibroma of the ovary in 1923 and found 2 cases with ascites and hydrothorax. Hydrothorax and ascites were found in a case of ovarian cyst by Wong[20] in 1928, and complete recovery followed surgical removal. Salmon[21] reported 2 cases of ascites and hydrothorax in 1934. One was an ovarian fibroma; the other was a fibromyoma of the uterus. Meigs[22] published his first observations on fibroma of the ovary associated with ascites and hydrothorax in 1934. The condition was mentioned another time in Cabot's case.[23] In 1937 Meigs[1] described the syndrome which bears his name. Besides the reports that have occurred in the literature since that date on fibroma of the ovary associated with ascites and hydrothorax,[2-9, 23] other reports[13, 16, 20, 21, 24] indicate that ascites and hydrothorax occur in association with other benign pelvic tumors of the uterus and ovaries. The important feature to be stressed is that the cases usually present a picture of hopeless malignancy, but when the benign tumor causing the symptoms is found and removed, there is a seemingly "miraculous" recovery. That death will occur if the tumors are not removed is

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evident from cases reported in the literature where the condition was not recognized.[2, 23]

The presence of a small amount of fluid in the peritoneal cavity, in association with cysts of the ovary on twisted pedicles and certain other strangulated tumors, is no doubt the knowledge of most surgeons who have encountered these conditions. But the presence of a large quantity of ascitic fluid in amounts of a gallon and more associated with a tumor of the ovary is indeed an apparently foreboding picture when the true nature of the tumor is not immediately known. It is most gratifying to see the change that occurs, as if by magic, when the benign tumor is removed and rapid recovery takes place. This is particularly true when hydrothorax has been present before surgery.

Case Report

Miss B. W., a white female, aged 55, was admitted to the hospital on 12 April 1947 with the chief complaint of enlargement of the abdomen of 3 months' duration.

The present illness had begun in December, 1946. At that time she had developed a digestive disturbance characterized by a fullness after meals, with bloating, belching, and eructation of food. Her appetite decreased because of the symptoms, and she began to lose substance in the flesh of her face, arms, legs, and chest. Weakness developed and was associated with some shortness of breath. As this developed, it was noted by her brother, who is a physician, that the abdomen was becoming larger. She had always been considered slightly neurotic, and no investigation into the complaints was effected until 24 Feb. 1947, when it was obvious that there was a real illness.

The family history revealed several close relations, one a brother, who had died of tuberculosis. The past history was entirely negative and irrelevant. She had passed the menopause at 45 years of age.

She consulted her local physician, who examined her carefully and completely. The only positive physical finding was "moderate general enlargement of the abdomen—tympanitic." Urinalysis gave negative findings. The red blood cell count was 3,570,000; the value for hemoglobin was 70 per cent, or 12 Gm. per 100 c.c.; the white blood cell count was 8,000, with 73 per cent polymorphonuclears, 26 per cent small lymphocytes, and 1 per cent eosinophiles. The gastric analysis with the Ewald test meal was normal. Roentgenograms of the chest, lungs, heart, esophagus, gallbladder, stomach, intestines, colon, and skeleton resulted in negative findings. The chest contained no fluid.

Several diagnoses were considered: cirrhosis of the liver, tuberculosis of the peritoneum, abdominal malignacy, origin unknown. It was decided laparotomy should be performed for diagnosis and she was admitted to the hospital for that purpose.

In the operating room under spinal anesthesia, the surgeon palpated the abdomen prior to making the incision, and under the complete relaxation of the spinal anesthetic, a large movable tumor was felt "floating" around in the several gallons of ascitic fluid. A low midline incision was made. Clear,

yellow fluid, estimated at more than 2 gallons, was aspirated from the abdominal cavity. Palpation and inspection of the liver revealed it to be normal. The other abdominal viscera were normal. There was a large nodular, hard, fibrous tumor of the left ovary, measuring 12 by 10 by 10 cm. The right ovary was normal. The peritoneum near the tumor was reddened and slightly granular, indicating the effects of an irritant which was thought to be from the tumor of the left ovary. There was nothing to indicate the pedicle was strangulated at this time or ever had been. The tumor was a fibroma.

Both ovaries were removed (figs. 1 and 2).

Convalescence was rapid and uneventful. There has been no recurrence of the fluid. Recovery is complete.

Incidence

For a very comprehensive study of Meigs' syndrome, reference is made to the abridgement of a thesis by Calmenson[16] who has made a complete review of the literature, as well as a study of some 20,000 cases of pelvic tumors. The study covered the 35 year period from 1910 to 1945 on tumors removed at the Mayo Clinic. He found the incidence of Meigs' syndrome to be 0.05 per cent. There were 9 cases of ascites and hydrothorax in the presence of pelvic tumors in the 20,000 cases studied, which included 500 ovarian fibromas. It is apparent that considerable difference of opinion exists as to the frequency of ascites and hydrothorax in the presence of fibroma of the ovary. The figure varies from 6 per cent by Rubin[17] to 40 per cent by Peterson.[24] It is safe to say that the figure of incidence of ascites in the presence of ovarian fibroma is about 5 per cent. Thus it appears that, while the occurrence of Meigs' syndrome is not rare, it is certainly not frequent. There are only 2 cases on record at the Mayo Clinic from 1937 to 1945.[16]

The age of incidence appears to be most marked in the fourth and fifth decades of life.

The chief complaint is nearly always enlargement of the abdomen with the associated pressure symptoms such as the sensation of bloating and difficulty in free breathing with dyspnea. There may be cough. Pain is an infrequent symptom.

The objective findings reveal ascites in all cases, which is usually of a rather marked degree. Hydrothorax is found in many cases but is not present in all. The pelvic tumor is not invariably discovered before surgery.

The diagnosis is not uniformly easy to make and many of the cases are misdiagnosed before operation as malignancy, tuberculosis, and cirrhosis of the liver. Other conditions to be considered in the diagnosis are cardiac and renal disease. In the case reported

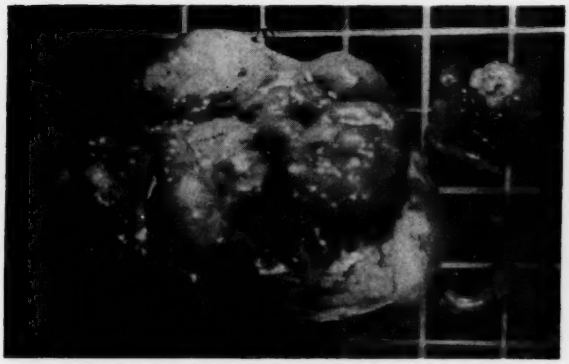

Fig. 1. Photograph of the tissue removed. The large tumor is a solid fibroma of the left ovary measuring 12 x 10 x 10 cm. The right ovary and the appendix are also in the photograph.

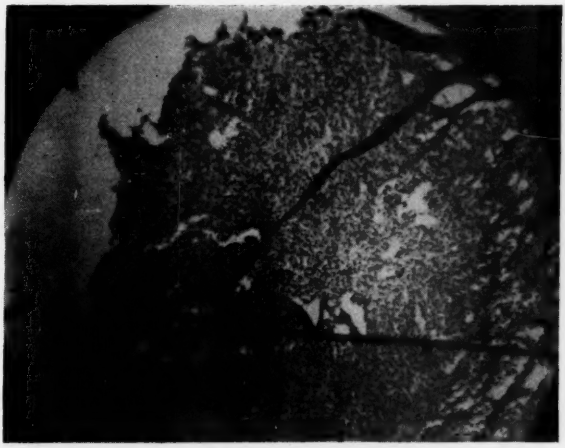

Fig. 2. Photomicrograph of stained section of tissue from the tumor of the left ovary, a fibroma. x 10.

in this paper, the preoperative diagnosis was malignancy of the ovary, and tuberculosis was not entirely excluded.

The types of tumor reported vary from fibromyoma to thecacell tumors, as well as fibroma, and cyst of the ovary.

The side of involvement is not constant.

The tumors encountered are nearly always of large size.

The amount of fluid present in the abdomen is usually reported as being of a large quantity and measures from 2 to 4 gallons.

There is nothing diagnostic about the character of the fluid. It is colloidal and clear and of straw color. The origin of this fluid is not clearly understood. There are several explanations offered, such as Selye's[25] "alarm reaction" to a damaging agent;[26] chemical reaction to an irritant; compression of lymphatics by the tumor with escape of this fluid into the peritoneal cavity; twisting of the pedicle of the tumor.[16] None of these explanations is entirely satisfactory. In the case reported in this paper, there is no doubt that an irritant was given off from the tumor as evidenced by the inflammatory reaction of the peritoneum near it. There was definitely no twisting of the tumor pedicle. There was never any pain to indicate it had ever twisted.

The cause of the hydrothorax is by direct passage of fluid through the diaphragm and this statement is based on Meigs' observations on 2 patients on whom he demonstrated that particulate matter passes from the abdominal cavity to the pleural cavity through the lymphatics of the diaphragm.

All cases of pelvic tumor with ascites do not exhibit hydrothorax. Rubin[17] has advanced four possible explanations. First, the fluid is absorbed by the pleura as fast as it arrives through the diaphragm. Second, in some cases the speed of flow of the fluid through the diaphragm is slowed down so that absorption occurs as fast as the fluid arrives in the chest. Third, the diaphragmatic channels become blocked and passage of the fluid is prevented. Fourth, and finally, the colloidal ascitic fluid produces a high osmotic pressure, which holds the fluid in the peritoneal cavity.

SUMMARY

1. Meigs' syndrome is briefly discussed.
2. A case of Meigs' syndrome without hydrothorax is presented.
3. Despite the foreboding clinical picture of inoperable malignancy, the condition responds promptly to extirpation of the benign tumor.
4. It is emphasized in reviewing the literature that death may occur if the pelvic tumor is not removed.
5. The occurrence of the disease is not common, and the clinician must be on the alert to detect it, keeping in mind that the pelvic tumor is not always discernible before surgery.

BIBLIOGRAPHY

1. Meigs, J. V., and Cass, J. W.: Fibroma of Ovary with Ascites and Hydrothorax, with Report of 7 Cases, Am. J. Obst. & Gynec. *33*:249-267, 1937.
2. Bomze, E. J., and Kirshbaum, J. D.: Fibroma of Ovary with Ascites and Hydro-

thorax; Clinical and Pathologic Study with Report of 2 Cases, Am. J. Obst. & Gynec. *40*:281-285 (Aug.) 1940.

3. Lock, F. R., and Collins, C. G.: Meigs' Syndrome: Fibroma of Ovary with Ascites and Hydrothorax, Am. J. Obst. & Gynec. *41*:517-519 (March) 1941.
4. Meigs, J. V.; Armstrong, S. H., and Hamilton, H. H.: Further Contribution to Syndrome of Fibroma of Ovary with Fluid in Abdomen and Chest, Meigs' Syndrome, Am. J. Obst. & Gynec. *46*:19-37 (July) 1943.
5. Herrick, W. W.; Tyson, T. L., and Watson, B. P.: Association of Hydrothorax with Ovarian Fibroma (Meigs' Syndrome), Arch. Int. Med. *71*:370-376 (March) 1943.
6. Clay, A. C.; Johnston, R. N., and Samson, L.: Meigs' Syndrome: Hydrothorax and Ascites in Association with Fibroma of the Ovary, British M. J. *2*:113-114 (July 22) 1944.
7. Gardiner, R. H., and Lloyd-Hart, V.: Ovarian Fibroma with Ascites and Hydrothorax (Meigs' Syndrome), The Lancet *2*:500-502 (Oct. 14) 1944.
8. Dockerty, M. B., and Masson, J. C.: Ovarian Fibroma; Clinical and Pathologic Study of 283 Cases, Am. J. Obst. & Gynec. *47*:741-752 (June) 1944.
9. Schaffner, V. D., and Kirkpatrick, T. A.: Pleural Effusion Associated with Ovarian Tumors (Meigs' Syndrome), Canadian M. A. J. *55*:55-57 (July) 1946.
10. Cullingsworth, C. J.: Fibroma of Both Ovaries, Tr. Obst. Soc., London (1879), *21*:276-288, 1880.
11. Tait, L.: On the Occurrence of Pleural Effusion in Association with Diseases of the Abdomen, Med.-Chir. Tr. Lond. *75*:109-118, 1891-2.
12. Mapes, C. C.: Ovarian Fibromata, Internat. Clin. *4*:205-227, 1909.
13. Kelly, H. A., and Cullen, T. S.: Myomata of the Uterus, Philadelphia, W. B. Saunders & Co., 1909, pp. 30-38.
14. Titus, R. S.: Fibroma of the Ovary Weighing Thirty-five Pounds; Operation; Recovery, Boston M. & S. J. *169*:381-384, 1913.
15. Caro, E.: Zur Lehre von den Riesenfibromen des ovariums nebst Bericht sines falles, in welchem ein 33½ Pfund schweres ovarialfibrom mit Erfolg exstirpert wurde, F. Lehmann, Leipzig, 1914, cited by Calmenson et al[16] from Rubin et al.[17]
16. Calmenson, M.; Dockerty, M. B., and Bianco, J. J.: Certain Pelvic Tumors Associated with Ascites and Hydrothorax, Surg., Gynec. & Obst. *84*:181-191 (Feb.) 1947.
17. Rubin, I. C.; Novak, J., and Squire, J. J.: Ovarian Fibromas with Thecacell Tumors; Report of Seventy-eight Cases with Special Reference to Production of Ascites and Hydrothorax (Meigs' Snydrome), Am. J. Obst. & Gynec. *48*:601-616 (Nov.) 1944.
18. Owen, A. W.: Fibroma of Ovary, Lancet, London *1*:1211-1212, 1923, cited by Bomze et al.[2]
19. Hoon, M. R.: Fibromata of Ovary, Surg., Gynec. & Obst. *36*:247-251, 1923.
20. Wong, A. I. H.: Ovarian Cysts with Torsion of Pedicle; Report of Fifty-two Cases from Gynecological Service of Johns Hopkins Hospital and Eleven Cases Observed in China, China M. J. *42*:625-645, 1928, cited by Calmenson et al.[16]
21. Salmon, U. J.: Benign Pelvic Tumors Associated with Ascites and Pleural Effusion, J. Mt. Sinai Hosp. *1*:169-172, 1934, cited by Calmenson et al.[16]
22. Meigs, J. V.: Tumors of the Female Pelvic Organs, New York, The Macmillan Co., 1934.
23. Meigs, J. V.: Fibroma of Ovary with Ascites and Hydrothorax; Further Report, Ann. Surg. *110*:731-754, 1939.
24. Peterson, R.: A Consideration of Ovarian Fibromata Based on a Study of Two Recent Cases and Eigty-two Collected from the Literature, Tr. Am. Gynec. Soc. *27*:264-295, 1902, cited by Calmenson et al.[16]
25. Selye, H.: Syndrome Produced by Diverse Nocuous Agents, Nature, London, *138*:32, 1936, cited by Calmenson et al.[16]
26. Schenck, S. B., and Eis, B. M.: Papillary Cystadenocarcinoma of Ovary with Hydrothorax, Am. J. Obst. & Gynec. *38*:327-330, 1939.

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CANCER OF THE STOMACH

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CANCER in all its manifestations is the greatest problem confronting scientific medicine today. It is second only to cardiorenal disease as a cause of death in individuals past middle life and 25 per cent of these cancer deaths are attributable to cancer of the stomach. The disease accounts for approximately 38,000 deaths each year, three fourths of them in men. The symptoms usually associated with cancer of the stomach might be better described as those indicative of an early demise and a rapidly approaching funeral rather than warnings of nature urging us to seek curative treatment.

Some clinics and medical centers where patients more favorable for treatment are seen report cures of cancer of the stomach in encouraging numbers, and perhaps some individual surgeons may find cause for optimism in the results of their own cases, but the report of Hinton and Abrahamson[1] from the Fourth Surgical Division of the Bellevue Hospital in New York probably accurately emphasizes the dismal picture presented by this disease. Of 444 patients with cancer of the stomach who entered the hospital, 296, or two thirds, were found to be inoperable on admission and of these 36 were moribund. Advanced age, extreme cachexia, lesions obviously too extensive on clinical examination for removal, evidence of distant metastases, such as jaundice, enlarged cervical nodes, involvement of the umbilicus, ascites, etc., making the outlook absolutely hopeless. After careful study 148 patients, or one third, were considered operable, and some improvement might be hoped for after surgical intervention. However, in 69 of these only exploration could be done and no further measures were taken, and of these 34 died soon after operation. In 55 cases palliative operations only could be performed, most of them for the relief of pyloric obstruction, and death ensued in all within a year. Only 24 cases of the 444, or 5.4 per cent of the total group and 16 per cent of the 148 operable cases, could be subjected to gastric resection. These figures probably represent the most pessimistic outlook, but better statistics can be obtained only in a more carefully selected group of patients.

The symptoms of cancer of the stomach are determined partly by the site of the growth, its extent and by the degree of motor impairment which it causes. A lesion high on the lesser curvature or one that lies on the anterior or posterior wall may be silent until well advanced. Indeed the discovery of metastases may precede the

finding of the parent growth. A tumor at the pylorus or near the cardiac orifice may cause obstruction early and thereby disclose its presence. There is great variability in the manifestations of this dreadful disease. The growth may simulate peptic ulcer and be considered benign, especially at the onset, and may even show evidence of temporary improvement under medical treatment, only to be followed by the substitution of an unremittent and continuous clinical course.

Usually however the presenting symptoms for which the patient consults a physician are epigastric discomfort or pain, present in about 90 per cent of cases, loss of weight, weakness, cachexia, and less frequently jaundice, nausea and vomiting and hematemesis. A palpable mass in the upper abdomen is present in about 60 per cent of the patients when first seen by a doctor. The duration of the symptoms frequently offers no relationship to the resectability or operability of the growth. The belief that cases of cancer in which the symptomatology is recent in development hold a better prognosis is in gastric cancer a fallacy. Reports of studies of series of cases actually show that the percentage of resectability and operability in groups in which the duration of symptoms was less than 3 months to be smaller than in those with symptoms of longer duration. Presumably, a cancer may be so malignant as to progress in 3 months too far to be amenable to treatment. It is obvious, therefore, that our criteria for diagnosis of early cancer of the stomach are faulty, and the symptomatology we depend on is absolutely unreliable for early diagnosis. In the great majority of instances it is only by accident that early and favorable cases come to operation.

Roentgenology is by far the most important laboratory aid to diagnosis and is accurate in over 90 per cent of the cases. Gastroscopy will show those small lesions which escape even the most experienced radiologist. However the disease is rarely suspected in a stage where the use of the gastroscope is essential to diagnosis. A study of the gastric contents will show hypoacidity in over 80 per cent of the patients with gastric cancer; but since hypoacidity is frequently found in normal individuals over 50 years old, the findings in this test are not diagnostic. However the absence or diminution of free hydrochloric acid must arouse strong suspicion and necessitates the positive exclusion of cancer of the stomach. Perhaps the development and use of new means of establishing the diagnosis in its earliest stages, or the discovery of the cause or causes of cancer, will increase the curability of this disease, but until such a time, improvement will only come from the constant alertness of the physician, to be always on his toes, to recognize the possibility of this disease and exclude it in every person in the

cancer age whose diagnosis is at all obscure. If there is any persistent disturbance of gastric function, mild or severe, especially if of recent origin, if the history reveals any irregular or atypical ulcer symptoms; if there is a departure from the normal in the gastric analysis, any tendency toward gastric retention or obstruction, the possibility of cancer must immediately come to mind and roentgenologic studies be carried out. Any progressive or unexplained anemia, loss of weight or strength, vomiting or anorexia, should immediately arouse the apprehension of the alert physician. Only by being constantly cancer-conscious can we make any progress in the fight against this disease. Then, too, the public should be informed of the importance of such symptoms so that patients will not delay seeking the advice of the physician. It is particularly discouraging that while the diagnosis can be made early, it very seldom is.

Concerning gastric malignancy there are two established and unexplained facts: the age incidence and the sex discrepancy. The great majority of patients with cancer of the stomach are men, and are over 50 years old. This has led to the postulation that there is an actual relationship between the sex hormone control of the gastric mucosa and gastric pathology including neoplastic changes. Perhaps further experimental work along this line of thought may throw some light on the problem of the etiology of gastric cancer. At present the cause or causes of gastric cancer, like that of cancer generally, is unknown. For many years there has been a wide divergence of views concerning the probability and frequency of malignant change occurring in chronic gastric ulcer. Some authorities believe that such changes occur quite often and consider any large gastric ulcer as potentially, if not actually, malignant. Others are equally certain that such a change is not only unusual but extremely uncommon.

The disease appears to be widely prevalent in certain families; the most famous is that of Napoleon I, who died of gastric cancer as also did his father, his brother and two of his sisters. There is also a wide racial discrepancy in the incidence of carcinoma of the stomach, for example it is quite frequent among the Japanese and five times as common in Chinese as in Malays living under the same conditions.

It has been pointed out that pathologically carcinoma of the stomach may be readily divided into two types: one, the type that more or less localizes in the stomach and rapidly metastasizes to the regional lymph nodes, and the other in which the cancerous infiltration spreads through the muscle bundles in the stomach wall,

often involving the entire organ, but not so rapidly metastasizing. In the stomach the blood and lymph channels form such a complex network that each part of the stomach has a vascular and lymphatic connection with every other part, and often tumor cells can be found present in the wall far beyond the limits of the grossly demonstrable tumor. Cancer of the stomach spreads to the regional lymph nodes, to neighboring organs and to distant parts of the body. However it is remarkable that it rarely spreads to the duodenum. The nearest nodes are always involved by the time the patient comes to operation.

In the treatment of gastric cancer it should always be remembered that death will certainly occur within a few months unless the course of the disease is interrupted. The purpose of treatment therefore is to cure the patient if possible or to prolong life and reduce suffering to a minimum if cure is hopeless. Non surgical methods of treatment including radiation are only palliative at best. It is a fundamental principle that every patient with gastric carcinoma should be given the benefit of an exploratory operation, unless it is obvious that even a palliative procedure is unwise. Fortunately this can often be determined. Massive involvement of the liver, with or without jaundice, a fixed mass, metastases in the umbilicus or ascites, when demonstrable clinically, contraindicate operation regardless of other circumstances. Palliative measures are usually directed toward relieving pyloric obstruction, and are indicated although the condition is known to be incurable. As in the surgical treatment of cancer elsewhere, so in cancer of the stomach, the more widespread the removal of the tumor and the adjacent tissues to which it may spread, the better the patient's chance of cure. When exploring for cancer of the stomach it is of utmost importance that the examination be thorough. The extent of the extra-gastric involvement should be determined. In addition to the more obvious sites, the peritoneum, the rectal shelf, omentum and the lesser peritoneal cavity should be investigated for the presence of secondary growths. Several factors determine the removability of the lesion. A short heavy patient makes any type of gastric operation difficult. If the stomach is small and lies high up under the diaphragm, an infiltrating growth may be clearly inoperable, while a similar growth in a prolapsed stomach may be easily removed. Improvement in the results of the surgical treatment of cancer has always followed increasing radical operations, so removable metastases in the omentum, spleen, pancreas, liver or mesentery should be included in the attack on the stomach itself.

The tremendous advances in recent years in the fields of anesthesia and nutrition, the routine employment of adequate and careful pre-

operative preparation, restoring the patient to the best possible condition before operation; the free use of blood, blood plasma and other blood substitutes, the truly miraculous benefits derived from the use of the sulfonamides, the antibiotics, and the parenteral use of protein digests, have made safe far more extensive procedures for cancer of the stomach than was formerly thought possible, and have created a changing concept concerning the attack upon the disease. As far as general principles are concerned, the growth should be removed as widely as possible; gastrointestinal continuity should be re-established without undue tension and the mechanical function should be perfect. Logically this ideal should conform to that in the treatment of carcinoma elsewhere, such as in the breast where the radical removal of the entire organ together with all anatomically related nodes and contiguous structures is done in block. Undoubtedly the block excision of the stomach and regional nodes together with the spleen and greater omentum should be the most effective means of treating gastric cancer. In the past total gastrectomy has been reserved for the treatment of those malignant lesions which could not be locally removed by subtotal resection. Since it is possible to perform total gastrectomy with a mortality rate approaching that following less extensive removal, and since there is sufficient evidence that gastrectomized patients maintain a fairly satisfactory nutritional state, the use of the procedure should be extended to the treatment of less extensive growths of the pyloric and fundic regions of the stomach, in those patients whose general condition and life expectancy make it feasible. Total gastrectomy has been done often enough and by a sufficient number of surgeons to prove there is no obvious reason why one should not live indefinitely without a stomach. While the mortality rate is and will probably remain higher for total gastrectomy than for subtotal resection, this may be somewhat discounted by the fact that in the past the more radical procedure has been done on the more advanced cases and often as a forlorn hope. With the more frequent use, greater experience and the more favorable selection of cases for the operation, there is reason to hope that the improved morbidity after total gastrectomy should more than offset the increase in mortality.

Connor[2] in 1884 reported the first total gastrectomy and since then more than 300 cases have been reported in the literature. Reports of fairly large series have been published by Finney and Rienhoff,[4] Waugh and Fahlund,[8] Lahey and Marshall,[5] Farris, Ransom and Coller,[3] Longmire,[6] Smith[7] and others. These reports lend encouragement to the belief that better results in the treatment of gastric cancer might be expected if the operation were more

frequently employed, especially in the treatment of the less extensive lesions in younger subjects. If this is done every effort must be made to lower the operative risk and reduce the postoperative complications to a minimum.

The establishment of a satisfactory postoperative nutritional program is essential. Studies of the digestive function in patients who have survived total gastrectomy as long as 3 years indicate they are able to lead an active life with few dietary restrictions. Most of these patients have gained some weight, all have good appetites and eat well, but the meals are usually smaller than those eaten by the normal person, and more frequent feeding is advisable. In the occasional case the jejunal pouch which substitutes for a stomach dilates so that the individual may be active on only three meals a day. Patients who have recovered from total gastrectomy have resumed a major portion of their usual activity although the return to normal muscular strength is slow. Some complain of epigastric fulness and burning after eating. In this they are not unlike some of the patients who have recovered from sub-total removal of the stomach and this complaint can usually be controlled by the administration of amphogel or some similar product.

Farris, Ransom and Coller studied the gastric function of several patients who had survived total gastrectomy for over 4 years. They found no evidence of disturbance in the digestion of fats and proteins. The absorption of glucose was more rapid than normal. The chief disturbance noted was in the metabolism of iron. In the absence of hydrochloric acid from the stomach the ferric and organic iron in the diet were not absorbed, and hypochromic microcytic anemia developed unless the patients were given ferrous iron regularly. Other investigators have found patients recovered from total gastrectomy in whom the digestion of fats and proteins was impaired, and there are some patients who apparently have been unable to adjust their digestive apparatus to the absence of a stomach and who died as a direct result. We must remember however that we are dealing with cancer of the stomach and all the patients had impaired digestive function of a great degree and were doomed to a certain death anyway. The majority of those who have died after total gastrectomy, did so as the result of recurrence of cancer. The average duration of life of those who survive total gastrectomy is 2 years. Waugh reported 2 patients who underwent total gastrectomy for benign lesions who are alive and well 6 and 8 years respectively after operation.

There has been a remarkable reduction in the mortality of gastric surgery, in fact surgery in general, attributable to better anesthesia

and anesthetists, more thoughtful preoperative preparation of the patient, increasing experience of surgeons dealing with this type of disease, the improvement in postoperative care and the early recognition and vigorous treatment of postoperative complications.

Our stomach cases (ulcer and cancer) are hospitalized for 3 to 7 days before operation and every effort is made to restore their nutritional balance if possible, replace blood loss, destroy latent infection, and promote physical rest. We assume, and believe correctly so, that anyone who has a serious stomach lesion can't help being in a chronic deficiency state, regardless of the laboratory findings. If stomach retention is present, constant suction is instituted and every effort made to get the stomach clean. Relief of the retention will reduce the edema of the stomach wall and often permits the administration of fluids orally in a few days. One or several whole blood transfusions are given. In addition fluid balance is restored by giving 3000 c.c. parenterally in 24 hours. This is usually made up of 1000 c.c. of 10 per cent glucose in normal saline solution, 1000 c.c. of 10 per cent glucose in water, and 1000 c.c. of a protein digest in 5 per cent glucose in water or normal saline solution, depending upon the degree of chloride loss by vomiting or gastric suction. One or two ampules of soluble vitamin B complex (Solu B. 5 X preferably) and 500 mg. of vitamin C are given daily in the intravenous fluids. Penicillin is given routinely. These patients have a low resistance to infection generally and lesions in the stomach are always potentially infected. Attention is given to the colon and it should be empty on the day of operation since it or the mesocolon may be found involved in the cancer, and require resection. We follow no set rule as to when to operate and are guided by clinical experience and do so when we think the patient has reached maximum improvement and is "about ready."

We recognize the highly important part the anesthesia and the anesthetist play in the successful management of gastric surgery. These patients as a rule are poorer operative risks than the average, are not infrequently advanced in years and often have other organic deficiencies besides the gastric lesion. We think one should use the anesthetic regimen that has been found satisfactory, and in our hospital we have found the combination of spinal and intravenous anesthesia more nearly approaches satisfaction than any other form of anesthesia we have used. We use a balanced anesthesia, which consists of the administration of sodium pentobarbital (nembutal) gr. 1½ about 2 hours before operation: morphine sulphate gr. 1/8 or gr. 1/6 and scopolomine or atropine gr. 1/150 an hour preceding going to the operating room. The dosage is varied somewhat depending upon the age, weight and general condition of the patient.

Procaine and pontocain mixed are used as anesthetic agents. Customarily we give 100 mg. of procaine and 12 to 14 mg. of pontocain dissolved in 5 c.c. of spinal fluid. The spinal tap is made in the interspace between the first and second or second and third lumbar vertebrae, depending upon the physical type of patient (tall or short, thin or fat) to whom it is given. This usually provides anesthesia and relaxation for two and a half hours which is adequate for nearly all of our resections, even the total. The spinal anesthesia is supplemented with 1 per cent sodium pentothal solution given intravenously, and only sufficiently to produce unconsciousness and prevent vomiting during the operation. It also relieves the patient of the trying ordeal of being awake, and in addition appears to have eliminated the dread and fear so many patients have of spinal anesthesia and the tendency to attribute everything that ever happens to them afterwards to the spinal tap.

Every effort is made to prevent or correct a prolonged fall of the blood pressure. Ephedrine sulphate in doses of 25 mg. to 50 mg. depending upon the diastolic pressure is used. If the diastolic pressure is over 100 mm. of mercury the smaller dose is given, and the larger dose is used in those patients whose diastolic pressure is less than 100 mm. of mercury.

Intravenous fluids, glucose solution, blood or blood plasma are used freely as needed during the operation and nasal oxygen is given continuously. Any sluggishness of respiration is controlled by administering nitrous oxide and oxygen by means of a gas machine with which positive pressure can be applied. Curare in doses of from 40 to 80 units, is occasionally used to increase the relaxation of the abdominal wall when closing the incision. The patient's lungs are hyperventilated with carbon dioxide and oxygen mixture, and secretions in the trachea are removed, as a routine measure before the patient leaves the operating room.

The operative technic is as follows. The abdomen is opened through an incision extending from the xiphoid process to the umbilicus, care being taken to go through the midline without exposing either rectus muscle. This gives ample exposure for all types of operations on the stomach, and for the removal of the spleen, transverse colon, portions of the pancreas and for the removal of metastatic nodules from the left lobe of the liver. It is our impression that this wound is less painful and definitely less embarrassing to respiration than the usual right or left rectus incision. The abdomen is carefully explored for metastasis and the extent and fixation of the growth determined. The situation is evaluated and the extent of the resection decided upon. We usually include the greater omentum in the removal and this is separated from the colon as

the first step. The lesser peritoneal cavity and the posterior wall of the stomach can then be visualized. The duodenum is divided and the stump closed and covered. The spleen is next mobilized and removed if it is to be included with the resection, if not the vasa brevia are ligated and divided and the stomach turned upward and to the left so as to expose the esophageal area. The left gastric artery and veins are divided as far from the stomach as possible and the lymph nodes in this region removed. The first loop of jejunum is brought through the mesocolon or in front of the colon as is considered best in the particular case, and anastomosed to the remnant of the stomach or to the esophagus as the case demands. We use an outer layer of interrupted silk or cotton sutures and an inner layer of chromic catgut, continuous for subtotal resections and interrupted if all the stomach is removed. In the latter procedure care is taken to cover the anastomosis with a peritoneal flap fashioned from the gastrohepatic omentum and peritoneum when the left gastric vessels are dealt with and the esophagus mobilized. We prefer to join the two loops of jejunum with an entero-anastomosis opposite the ligament of Trietz, and believe it makes for a more comfortable convalescence, prevents back pressure in the duodenum and enables the new stoma to function better. We do not hesitate to attempt the removal of any metastases that can be excised, whether in the mesentery, pancreas, liver or elsewhere. The incision is closed in layers using catgut for the peritoneum and fine stainless steel wire for the fascia, and drains are very seldom used. The midline incision permits rapid opening and closing of the abdomen with a minimum loss of blood.

Every effort is made to prevent complications after operation, and to discover them promptly and deal with them vigorously when they occur. We are especially watchful for atelectasis, thrombophlebitis, parotitis, renal failure and cardiac strain. Most of our patients, and all of the elderly ones, are placed in an oxygen tent immediately, and hyperventilation of the lungs with carbon dioxide and oxygen carried out hourly while the patient is awake for the first 48 hours after operation. If atelectasis develops we try to open the lung by having the patient cough, breathe deeply, turn on the opposite side, and by heavy percussion over the affected lung area. We resort to bronchoscopic aspiration when necessary. We encourage the patients to turn freely and exercise in bed and not infrequently get them out of bed on the third day. We try to exercise judgment in the use of early ambulation. Special attention is given the care of the mouth, acid lozenges are prescribed and gum may be chewed if the patient has teeth. Penicillin and sulfonamides are continued postoperatively.

Nothing is of greater importance in the postoperative care of these patients than maintaining the nutrition during the period of time when oral feeding is withheld. The routine administration of protein digest by vein in amounts equal to 50 to 100 grams of protein daily during this time is of prime importance in the promotion of wound healing, resistance to infection and the prevention of edema. The maintenance of the caloric intake is also essential. This is provided by the liberal use of glucose. We find a daily regimen of 1000 c.c. of protein digest in 5 per cent glucose solution, 1000 c.c. of 10 per cent glucose solution to which the soluble vitamin B complex is added in large doses, together with the administration of 300 mg. of vitamin C will tide the patient over for the required length of time until oral feeding can be resumed. This provides approximately 1200 calories, the equivalent of 50 grams of protein and 8.5 grams of sodium chloride.

In subtotal gastrectomy patients, water, tea with milk and sugar, Coca-Cola, etc., in one ounce amounts per hour are permitted on the second day and, if well tolerated, fluids are increased rapidly. If the stomach empties well, soft foods such as cooked cereal, soft eggs, custard, ice cream, vegetable purees and scraped beef are given in small quantities at two hour intervals. We encourage these patients to eat and get them on a high caloric diet as early as possible, as much as 3500 to 5000 calories a day by the time they leave the hospital.

Patients who have undergone total gastrectomy are given nothing by mouth for 3 days. On the fourth day an ounce of water every hour is permitted. On the following (fifth) day an ounce of peptonized milk or malted milk, cream soup or ovaltine is given with the water at half hour intervals. The amount of fluid is increased gradually and by the seventh or eighth day, soft eggs, custard, cooked cereal, skimmed milk powder is added and, as rapidly as tolerated, both the quantity and the caloric intake are increased. The parenteral feeding as previously outlined is continued until the patient is taking a maintenance diet by mouth, consisting of 6 feedings a day of a high protein, high carbohydrate, low fat and low residue diet. The effort is made to increase the diet by 300 calories a week. The jejunal pouch which substitutes for a stomach gradually adapts itself so that in time the patient is able to maintain weight and strength on 6 or less meals a day.

It should be emphasized that patients with gastric cancer are in a state of chronic dietary deficiency, and failure to realize this and neglect in the recognition of the extreme importance in dealing with this factor both before and after operation will contribute to

an undue mortality rate, regardless of the technical perfection with which the operative procedure is accomplished.

Analysis of Cases

Number of operated cases—32
- Males—27
- Females—5

Pathologic Diagnosis:
- Lymphosarcoma—3
- Linitis Plastica—2
- Carcinomatous Ulcer—3
- Adenocarcinoma—24

Operative Procedure:
- Gastroenterostomy—2
- Closure of Perforation—1
- Exploration—Biopsy—5
- Total Gastrectomy—4
- Resection in two stages—1
- Resection in presence of metastases—2
- Subtotal Gastrectomy—17

Hospital Deaths—7
- Before use of sulfonamides—5
- Since use of sulfonamides—2

Cause of Hospital Death:
- Pneumonia following unrecognized atelectasis. (Total Gastrectomy)
- Streptococcic empyema complicating streptococcic sore throat. (Subtotal Gastrectomy—Lymphosarcoma)
- Exhaustion and cardiorenal failure—10th day—Age 78. (Subtotal Gastrectomy—Adenocarcinoma)
- Peritonitis from accidental leakage following enema. (Subtotal Gastrectomy—Antecolic Anastomosis—Adenocarcinoma)
- Parotitis—bilateral—fulminating. (Total Gastrectomy—Linitis Plastica)
- Pulmonary Embolus—4th night following operation. (Subtotal Gastrectomy—Adenocarcinoma)
- Postoperative Shock—following attempt to resect pancreas. (Gastroenterostomy—Adenocarcinoma)

Died since leaving hospital—11
- Died with cancer—10
- Died without cancer—1
  (Accident 5 years after operation—two stage resection—Adenocarcinoma)

Living—14
- Living with known recurrence—4
- Living without recurrence—9
- Living when lost sight of—1

Living longer than 3 years following operation—4

Pathologic Diagnosis of those living longer than 3 years—
- Carcinomatous ulcer—3 (19 years—6 years—7 years)
- Adenocarcinoma—1 (Patient who died in accident)

SUMMARY

Cancer of the stomach is a most discouraging problem since our diagnostic criteria rarely permit the discovery of the disease when the lesion is pathologically young and amenable to treatment. Until new methods of diagnosis are available there is little to stimulate optimism for any improvement in the future. It is recommended that every physician be constantly cancer-conscious and especially on the alert to recognize symptoms suggesting cancer of the stomach, and the more frequent use of roentgenology in examining such patients is advised. Surgery is the only treatment that offers any hope of arresting the course of the disease, yet it is purely accidental when patients come to operation in a stage where cure is possible. Since total gastrectomy more nearly meets the ideal concept of the surgical attack on cancer, it is suggested that this procedure be more frequently employed in preference to the less extensive gastric resections, in selected cases. Emphasis is placed on the great importance of preoperative preparation, anesthesia and postoperative care in the management of these poor risk patients. Analysis of 32 operative cases is presented. The apparent cure of the 3 patients with carcinomatous ulcer indicates the comparatively low degree of malignancy of this type of lesion.

REFERENCES

1. Abrahamson, R. H., and Hinton, J. W.: Carcinoma of Stomach; Review of 444 Cases to Emphasize Inadequacy of Present Methods for Early Diagnosis, Surg., Gynec. & Obst. *71*:135-141, 1940.
2. Connor, P. S.: Discussion, Med. News, New York *45*:578, 1884.
3. Farris, J. M.; Ransom, H. K., and Coller, F. A.: Total Gastrectomy; Effects upon Nutrition and Hematopoiesis, Surgery *13*:823-833 (June) 1943.
4. Finney, J. M. T., and Rienhoff, W. F., Jr.: Gastroectomy, Arch. Surg. *18*:140-162, 1929.
5. Lahey, F. H., and Marshall, S. F.: Symposium on Abdominal Surgery. Indications for, and Experiences with, Total Gastrectomy; Based upon Seventy-three Cases of Total Gastrectomy, Ann. Surg. *119*:300-320 (March) 1944.
6. Longmire, W. P., Jr.: Total Gastrectomy for Carcinoma of Stomach, Surg., Gynec. & Obst. *84*:21-30 (Jan.) 1947.
7. Smith, F. H.: Total Gastrectomy; Report of 89 Cases, Surg., Gynec. & Obst. *84*:402-408 (April) 1947.
8. Waugh, J. M., and Fahlund, G. T. R.: Total Gastrectomy, S. Clin. North America *25*:903-917 (Aug.) 1945.

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YOUR SURGEON AND MINE

Occasionally a colleague is referred to as a great surgeon. Just what is meant by the term *great?* Is it the ability to perform some difficult surgical procedure that others dare not attempt? Is it some special talent that enables the particular surgeon to achieve better results with a lower mortality? Does he appear great because he works more swiftly and expertly, as the result of extensive experience? Is he a great anatomist, pathologist, and diagnostician?

All these faculties and others may be possessed by a surgeon, and yet he may not be great. For, regardless of skill and unusual technical ability, if he does not have good surgical judgment and, more especially, if he is not honest, he is not a great surgeon. If you and I were going to select a surgeon to operate upon a member of our family, honesty would be paramount among the qualities we would require of him. There might be surgeons who could work faster and who had achieved wide renown by virtue of large series of statistical successes, but they would not be our choice if we had occasion to doubt their honesty.

In this era of modern anesthesia, speed is no longer a vital factor in most surgical procedures. With less speed there would be fewer papers written about repair of the common duct, transplantation of

the ureter, and King's operation for paralysis of the recurrent laryngeal nerves. No, speed is not a vital requisite; rather, it is the end result that counts. It is the care and precision and attention to detail that reduces complications and improves end results.

Does boldness stamp a surgeon as great? Not necessarily. Boldness may be the result of ignorance—ignorance of the pitfalls that only time and experience teach one to avoid.

There is an old saying that statistics may be made to tell anything, even the truth, and so a surgeon may not be judged great just because of the fact that he publishes large series of cases. I have one series of cases that continues to grow, and I am not proud of it. This is a rather unique series of more than 200 patients who were advised to have a thyroidectomy performed for what seemed to me to be a normal thyroid gland. In only one instance had the thyroid been removed by a bold surgeon, and this unfortunate patient now must always take thyroid to prevent myxedema. Anyone may make an occasional mistake in the diagnosis of hyperthyroidism. Probably one who sees a considerable number of cases will err on occasion. But to mistake the condition often, or even infrequently, is a serious indictment of a surgeon's diagnostic acumen, if not of his honesty.

In 1937 I published a paper entitled "Needless Thyroid Surgery," a study of 100 cases. Among those who became interested in this subject was Dr. Elexious T. Bell, pathologist of the University of Minnesota Medical School. At his request this paper was presented before the Hennipen County Medical Society, and a group of 100 cases of normal thyroid glands was also shown by Doctor Bell. When one stops to consider that in the comparatively limited experience of one surgeon and one pathologist 200 apparently needless thyroidectomies were either advised or performed, the great number of uncalled-for operations being done is apparent. In an address to the American College of Surgeons at the New York meeting, Dr. Harold Foss called attention to the large number of operations being performed without justification. The lay press and the radio have brought this before the public eye. To this I can see no objection. Syphilis was whispered about and kept in a dark closet until it became so prevalent that it had to be brought out in the open where everyone could learn something about it.

It is not only innocent thyroid glands that are being removed without provocation. Dr. Norman F. Miller of Ann Arbor has done splendid work in recent years in calling attention to the great amount of unwarranted pelvic surgery and the resulting harmful sequelae. That, of course, is an old story; scarcely a day goes by

that I do not see a pathetic, neurotic, oophorectomized victim of someone's surgical zeal. Possibly in time the American Board of Surgery will be able to eliminate such surgery. In the better type of hospital such steps have already been taken by the staff itself. Too frequently this injudicious surgery occurs in the smaller institution that cannot be reached by the Board, the American Medical Association, or the American College of Surgeons.

There once lived a surgeon who was known as the surgeon's surgeon, because so many surgeons wanted only him to operate upon them when they were in trouble. He had great technical skill, and, while he was a rapid operator in an era that called for speed, he seldom made a false or purposeless move, so that he never gave the impression of being in a hurry. His touch was so deft, and he handled tissues with such extreme gentleness that there was always a minimum of trauma. Dr. William Mayo thought him the greatest surgeon he had ever seen. Yet what drew so many to him was not so much his remarkable skill as his sincerity and modesty. When Starr Judd said, "Maybe you better be operated upon," you were sure there was a good reason for that operation.

ARNOLD S. JACKSON, M.D.

REFERENCE

Jackson, A. S.: Needless Thyroid Surgery; an Analysis of One Hundred Cases, West. J. Surg. *45*:448-452 (Aug.) 1937.

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